Effect of Sugarcane Mosaic Virus Infection in Parental Stock on Panicle and Seed Production of Virus-Free F_2 Progeny in Sorghum (*Sorghum bicolor*)

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ABSTRACT

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Virus-free F_2 progeny of the original sorghum cross MN 4611 × Planter were analyzed to compare panicle and seed production among populations derived from parental stock infected with sugarcane mosaic virus strain A (SCMV-A) or not infected. Virus-infected crosses had only one parent infected with SCMV-A, either female or male. The virus was not present in F_1 or F_2 plants. Data were collected on 441 F_2 plants in greenhouse tests. Analysis of variance and LSD comparisons showed a significant reduction ($P = 0.005$) in seeds per panicle and an increase in panicles per plant ($P = 0.070$) of F_2 progeny derived from crosses with SCMV-A-infected MN 4611 compared with F_2 data from uninfected crosses. Our data suggest that there is varietal variation with respect to panicle and seed production in the F_2 in response to virus infection in the original crosses. Total number of seeds per plant did not vary significantly between F_2 progeny of uninfected vs. virus-infected groups because of the long period of plant growth (7 mo).

Additional key words: genetic deviation, yield components

Reduction of seed yield in virus-infected host plants has been shown for numerous host-virus combinations (1,6,8–10,15). Barnett and Gibson (1) reported seed yields of virus-free white clover were four times those of virus-infected plants. They found significant decreases in the number of seeds per head and per floret, weight of individual seeds, and number of heads per plant. Inouye (6) attributed sterility in barley plants infected with barley stripe mosaic virus (BSMV) to abnormal pollen and anthers. Slack et al (17) showed that the number of pollen grains per anther and the number of viable pollen grains in barley were diminished by BSMV. Sandfaer (15) found that although there is considerable varietal variation, BSMV increases the frequency of triploid and aneuploid seed production in barley. Plants produced by triploid and aneuploid seed have an increased number of sterile flowers. Sweet corn plants infected with maize dwarf mosaic virus strain A or B (MDMV-A or MDMV-B), a virus closely related to sugarcane mosaic virus (SCMV), have shown delayed maturity and poor ear quality because fewer kernels formed at the basal portion of the ear (9). SCMV has been reported in sorghum-growing areas worldwide. In field trials, significant seed reductions have been demonstrated in SCMV-infected sorghum plants (4,11,12,16). Henzell et al (5) found that decreases in grain weight and number of grains per panicle in SCMV-infected sorghum were influenced by varieties. MDMV reduced yields of grain per panicle by 84–86% in some sorghum varieties (20).

Much less is known about the effects of viruses in the virus-free progeny of crosses involving virus-infected parents. Previous reports have attributed genetic deviations in virus-free plants to virus infection in their parental stock. Sprague and McKinney (18), working with BSMV-infected maize, assigned the term "aberrant ratio" (AR) to a phenomenon associated with virus infection of parent plants. Virus-free F_2 and backcross segregation ratios showed consistent and significant departures from Mendelian expectations. Samson et al (14) hypothesize that virus infection of the meristem or pollen precursor cells at some critical stage induces AR indirectly by interacting with a maize controlling element.

A previous report (19) demonstrated the mutagenic effect of SCMV strain A (SCMV-A) on sorghum manifested by significant departures from expected Mendelian segregation ratios in F_2 populations. Our study was undertaken during research to evaluate the F_3 segregation of that population. The observation of abnormal reproductive structures and poor seed production in F_2 seedheads prompted us to collect the data reported in this paper.

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MATERIALS AND METHODS

Data were collected from 441 F_2 plants derived from the cross MN 4611 × Planter, in which there were either two uninfected plants (controls) or one uninfected and one SCMV-A infected plant. The parents were MN 4611 (female), a selection from Day, a milo type sorghum, and Planter (male), a sweet sorghum. Virus-infected parent plants were obtained by mechanically inoculating seedlings, either male or female, with SCMV-A in the three-leaf stage. After panicle emergence, all female plants were hot water emasculated at 47 C for 10 min, then hand-pollinated for 2–3 days. All crosses were performed in a greenhouse within 1 mo. Greenhouse temperatures ranged from 27 to 30 C. F_1 plants, grown from seed obtained from the crosses, were self-pollinated to produce seed for F_2 plants. These seeds were planted at one time in randomly arranged 13-cm clay pots. F_2 plants were grown in the greenhouse for about 7 mo, during the spring to early fall of 1981. Greenhouse temperatures ranged from 27 to 38 C. Panicles of these F_2 plants were covered with glassine bags before anthesis to produce self-pollinated seed.

Plant height, measured from the soil line to the base of the peduncle, was recorded when the first panicle produced on a plant (main stem panicle) was mature. Self-pollinated panicles on the main stem and side branches of each F_2 plant were harvested at the end of the growing period. All panicles of a plant were placed together for drying, the mature seed heads were threshed, and the seed were pooled for each plant separately. The following yield components were evaluated: 1) panicles per plant; 2) seed per panicle, the number of seed per plant divided by the number of panicles with seed harvested from that plant; and 3) seed per plant. Because exact seed counts were not made for all plants, classes were set up with the class midpoint used for statistical analysis as follows: 0–10 seeds (midpoint = 5), 11–20 seeds (midpoint = 15), etc.

Data on height and yield components were subjected to an analysis of variance and a test of least significant difference (LSD). Comparisons were made between the data from F_2 plants derived from the following original crosses: uninfected vs. virus-infected, in which the female (MN 4611) was infected with SCMV-A, and uninfected vs. virus-infected, in which the male (Planter) was infected with SCMV-A. To evaluate the differences in efficiency of the three population groups in panicle and seed production at different growth stages (side branch production), a 2 × 6 chi-square contingency table analysis was performed.

RESULTS AND DISCUSSION

Positive virus recovery attempts from random virus-infected parent plants correlated viral symptoms present with SCMV-A infection. Bioassays for SCMV-A in F_1 and F_2 plants produced by crosses with virus-infected parent plants were negative. This agrees with previous reports (7,13).

There were 158 F_2 plants in the uninfected cross group developed from five crosses and 28 F_1 plants. Three crosses in which MN 4611 female plants were infected with SCMV-A produced nine F_1 and 152 F_2 plants. Also, nine F_1 and 131 F_2 plants were derived from three crosses in which the Planter male plants were infected with SCMV-A.

Data from virus-free F_2 plants derived from crosses that had SCMV-A-infected MN 4611 as the female parent showed a highly significant reduction in the number of seeds per panicle ($P = 0.005$) and a 20% increase in the number of panicles per plant ($P = 0.070$) compared with data from F_2 plants from uninfected crosses (Table 1). When data from virus-free F_2 plants derived from crosses that had SCMV-A infected Planter as the male parent were similarly compared with the uninfected crosses, a reduction in number of seeds per panicle was significant at the $P = 0.068$ level of confidence. The 13% increase in the number of panicles per plant was not significant ($P = 0.239$) (Table 1). Plant height and the total number of seeds per plant were not significantly different among the F_2 data collected from the three groups of crosses.

We observed abnormal anthers in some F_2 plants. Among the abnormalities, the anthers were apparently normal in shape but indehiscent and reddish; lean, apparently starved and light yellow; or were degenerated into small dark lumps, nearly invisible or leaving naked filaments. These observations are similar to those in beet yellows virus-infected sugar beets reported by Larsen (8).

The increase in number of panicles per plant in the F_2 populations from virus-infected crosses is similar to results of previous work in sorghum and ryegrass. Broadhead (2) found that removing the main stem panicle of sweet sorghum plants before seed formation caused an increase in the formation of side branches. In our study, poor seed production in F_2 populations from virus-infected crosses had a similar effect on the production of side branches and panicles. A decrease in seed production on the main stem and first side branch panicles caused an increase in the number of side branch panicles formed. In work with ryegrass, Catherall and Wilkins (3) found that infection with barley yellow dwarf virus usually reduced the ratio of fertile to vegetative tillers. Infected plants tillered excessively so that the vegetative phase was prolonged, a response similar to the increased F_2 panicle production induced by SCMV-A in the crossing material of our study. The F_2 populations from each virus-infected cross group had plants producing a significantly greater number of panicles compared with the F_2 populations from uninfected crosses (Table 2).

Extending the growing period of F_2 progeny from uninfected and virus-infected crosses resulted in comparable yields of seed per plant among the three groups (Table 1). In commercial production of sorghum seed and the research reported earlier involving sorghum, plants were harvested and data recorded when seed on the main stem panicle and, possibly, the first side branch panicle were mature. Our data also suggest a decrease in production of seed in the F_2 progeny of virus-infected crosses when only seed on these two panicles are mature. As seen in Table 2, 50% of the F_2 plants from the uninfected group produced their full seed complement from the main stem and first side branch panicles, whereas only 35% (SCMV-A/MN 4611) and 40% (SCMV-A/Planter) of the plants from virus-infected parentage did so. So even though there was no significant difference in total production of seed per plant, a larger proportion of the F_2 population from uninfected crosses produced their seed on the first two panicles, whereas the F_2 populations from virus-infected crosses produced their seed on a greater number of panicles per plant (Fig. 1).

The influence of SCMV-A on seed production of sorghum reported in this paper indicates that although the virus is not seed-transmitted and is absent in F_2 plants, a significant effect of SCMV-A infection in the parents of the original cross is observed in the ability of the F_2 plants to produce panicles and seed. Our data suggest that the effect on panicle and seed production may be influenced by differences in virus/host combinations. Preliminary findings in the evaluation of segregation ratios of virus-free F_3 plants support the theory of different varietal responses to virus infection (I. E. Stokes, *unpublished*). Delay in seed production

Table 1. Analysis of height and panicle and seed production of virus-free F_2 plants derived from the cross MN 4611 × Planter in which parents were either uninfected or virus-infected[a]

	Parents		
		Virus-infected	
F_2	Uninfected	MN 4611 (female)	Planter (male)
Height (cm)	84.10[b]	72.20 (0.169)[c]	75.00 (0.295)
Panicles per plant (no.)	2.68	3.22 (0.070)	3.02 (0.239)
Seeds per panicle (no.)	16.78	13.31 (0.005)	14.79 (0.068)
Seeds per plant (no.)	30.13	27.04 (0.106)	27.29 (0.146)

[a] Either female or male plant infected with sugarcane mosaic virus strain A.
[b] Data are means for entire groups.
[c] Numbers in parentheses represent probability levels associated with differences from the corresponding uninfected plants as calculated by the method of least significant difference (LSD).

Table 2. The number and proportion of panicles produced by virus-free F_2 plants derived from the cross MN 4611 × Planter in which parents were either uninfected or virus-infected[a]

	Production of panicles on F_2 plants[b]						
		Main stem and side branch (total)					
Parents	Main stem only	2	3	4	5	6	Total F_2 plants (no.)
Uninfected							
No.	14	65	49	19	10	1	158
Freq. (%)	8.9	41.1	31.0	12.0	6.3	0.6	···
Virus-infected							
MN 4611[c] (female)							
No.	6	47	44	26	20	9	152
Freq. (%)	4.0	30.9	29.0	17.1	13.2	5.9	···
Planter[c] (male)							
No.	14	38	31	28	19	1	131
Freq. (%)	10.7	29.0	23.7	21.4	14.5	0.8	···

[a] Either female or male plants infected with sugarcane mosaic virus strain A.
[b] Fourteen plants produced only one panicle (main stem only); 65 plants produced two panicles (main stem and first side branch only), etc.
[c] Distribution of panicles of F_2 plants are significantly different at the $P = 0.004$ (uninfected vs. SCMV-A-infected MN 4611) and $P = 0.021$ (uninfected vs. SCMV-A-infected Planter) levels of probability based on chi-square 2 × 6 contingency table analyses.

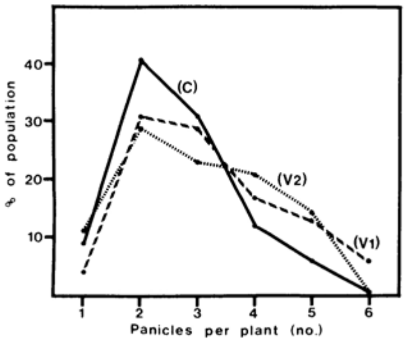

Fig. 1. Number of panicles produced per F_2 plant as influenced by the introduction of sugarcane mosaic virus strain A into selected parental plants. C = uninfected controls, V1 = virus-infected MN 4611, V2 = virus-infected Planter.

would be an important factor in a commercial program where harvesting is done when seed have matured on the main stem and first side branch panicles.

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LITERATURE CITED

1. Barnett, O. W., and Gibson, P. B. 1977. Effect of virus infection on flowering and seed production of the parental clones of Tillman white clover (*Trifolium repens*). Plant Dis. Rep. 61:203-207.
2. Broadhead, D. M. 1973. Effects of deheading on stalk yield and juice quality of 'Rio' sweet sorghum. Crop Sci. 13:395-396.
3. Catherall, P. L., and Wilkins, P. W. 1977. Barley yellow dwarf virus in relation to the breeding and assessment of herbage grasses for yield and uniformity. Euphytica 26:385-391.
4. Grancini, P., and Mariani, G. 1974. Sugarcane mosaic virus in sorghum. Mikrobiologija 11:9-17.
5. Henzell, R. G., Persley, D. M., Fletcher, D. S., Greber, R. S., and van Slobbe, L. 1979. The effect of sugarcane mosaic virus on the yield of eleven grain sorghum (*Sorghum bicolor*) cultivars. Aust. J. Exp. Agric. Anim. Husb. 19:225-232.
6. Inouye, T. 1962. Studies on barley stripe mosaic in Japan. Ber. Ohara Inst. Landwirtsch. Biol. Okayama Univ. 11:413-496.
7. Koike, H., and Gillaspie, A. G., Jr. 1976. Strain M, a new strain of sugarcane mosaic virus. Plant Dis. Rep. 60:50-54.
8. Larsen, K. 1981. Male sterility caused by beet yellows virus. Z. Pflanzenkr. Pflanzenschutz 87:111-115.
9. Mikel, M. A., D'Arcy, C. J., Rhodes, A. M., and Ford, R. E. 1981. Yield loss in sweet corn correlated with time of inoculation with maize dwarf mosaic virus. Plant Dis. 65:902-904.
10. Narayanasamy, P., and Jaganathan, T. 1975. Effect of black gram (mungbean) leaf crinkle virus infection on seed set and distribution of virus in the seeds. Madras Agric. J. 62(3):151-154.
11. Nour, A. H., Farrag, S. H., Hady, H. L. A., and Gohar, M. A. 1979. The effect of sugarcane mosaic virus on the growth and yield attributes of sugarcane and sorghum. Agric. Res. Rev. 57(8):11-21.
12. Persley, D. M. 1978. Sugarcane mosaic virus in sorghum. Queensl. Agric. J. 104(3):279-281.
13. Pirone, T. P. 1972. Sugarcane mosaic virus. Descriptions of Plant Viruses. No. 88. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England.
14. Samson, R. G., Brakke, M. K., and Compton, W. A. 1979. Evidence for gene inactivation in the virus-induced aberrant ratio phenomenon in maize. Genetics 92:1231-1239.
15. Sandfaer, J. 1974. Occurrence of triploids and aneuploids in BSMV-free and BSMV-infected barley material. Pages 289-294 in: Polyploidy and Induced Mutations in Plant Breeding. Proc. Symp. FAO/IAEA and EUCARPIA, Bari, Italy.
16. Shukla, K. 1981. Studies on yield loss of sorghum infected with sugarcane mosaic virus. Z. Pflanzenkr. Pflanzenschutz 88(10):635-637.
17. Slack, S. A., Shepherd, R. J., and Hall, D. H. 1975. Spread of seed-borne barley stripe mosaic virus and effects of the virus on barley in California. Phytopathology 65:1218-1223.
18. Sprague, G. F., and McKinney, H. H. 1966. Aberrant ratio: An anomaly in maize associated with virus infection. Genetics 54:1287-1296.
19. Stokes, I. E., Mock, R. G., Gillaspie, A. G., Jr., and Koch, E. J. 1982. The sugarcane mosaic virus as a mutagenic agent in sweet sorghum (*Sorghum bicolor* (L.) Moench). Sugarcane Pathol. Newsl. 28:35-41.
20. Villalon, B., and Creelman, R. A. 1981. Disease reactions of sorghum cultivars to inoculations with maize dwarf mosaic virus. Tex. Agric. Exp. Stn. 3913. 12 pp.