

## Transmission of Bean Pod Mottle Virus in Soybeans and Effects of Irregular Distribution of Infected Plants on Plant Yield

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### ABSTRACT

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Factors affecting bean pod mottle virus (BPMV) progression in soybeans and the effect of plants escaping virus infection on yield reduction were investigated. In 1980, 4 wk after inoculation with BPMV, rows of tall soybean genotypes (120 cm) adjacent to rows of relatively short genotypes (85 cm) had 35% more symptomatic plants than adjacent short lines. Differences became less as the season progressed. In 1982, tall lines (100 cm) next to short lines (78 cm) had a significantly higher number of BPMV-infected plants than adjacent short lines whereas no differences in BPMV disease incidence were found in adjacent soybean lines that were similar in

plant height. Disease incidence within soybean cultivars Ransom and Centennial planted on 25 June 1982 increased two to three times faster than within these cultivars planted on 11 May. Virus spread within rows appeared to be nonrandom at the beginning of the epidemic and became random as the epidemic progressed. Individual Centennial and Ransom soybean plants without virus symptoms adjacent to BPMV-infected-plants yielded 50 and 16% more, respectively, than did healthy plants adjacent to other healthy plants.

Immunity to bean pod mottle virus (BPMV) in soybeans (*Glycine max* (L.) Merr.) has not been found (7) but has been reported in certain species of *Glycine* (6). Symptoms of this disease range from a mild chlorotic mottle to a severe mosaic with the most obvious symptoms appearing on younger leaves. Soybean lines have been identified that are almost symptomless (5) or sustain little yield loss from this disease (8).

The bean leaf beetle (BLB) (*Cerotoma trifurcata* Forst.) is the primary vector of BPMV. Although soybean growth stage at inoculation with BPMV has been shown to influence foliar symptom expression (3) and yield losses resulting from infection (3,7), little or no research has been reported on the effect of either uninfected plants (escapes) in fields with the majority infected or of late-infected plants on the overall yield response to virus infection.

The objectives of this study were as follows: to determine the effect of time of planting and plant canopy height on disease progression; to determine the nature of BPMV spread within rows (random or nonrandom); and to determine the amount of yield compensation by apparently healthy plants when adjacent plants are infected with BPMV.

### MATERIALS AND METHODS

All field experiments were conducted at the Tidewater Research Station, Plymouth, NC, where the BLB was prevalent throughout the growing seasons. Planting dates were 19 May 1980, 19 May 1981, and 11 May 1982 unless stated otherwise; rate of planting was 21 seed per meter of row, and row width was 0.96 m. Mechanical inoculations were accomplished with a pad inoculator (4) containing soybean sap obtained from BPMV-infected soybean leaves diluted 1:10 (w/v) with 0.5 M phosphate buffer, pH 6.9; 0.25% Celite (Sigma Chem. Co., St. Louis, MO 63178) was added to the inoculum to serve as an abrasive.

The percentage of plants infected (disease incidence) was based on the percent of plants with chlorotic symptoms characteristic of BPMV infection. Symptom severity of infected soybean lines was determined by using the following assessment scale: 1 = mild chlorotic mottle on youngest leaf; 2 = mild chlorotic mottle on several of the younger leaves; 3 = chlorotic mottle to mosaic, some leaves crinkled; 4 = severe mosaic on top of canopy, older leaves crinkled; and 5 = severe mosaic on upper half of canopy, leaves crinkled. Scores were based on overall appearance of the plant row.

**Plant height effects on disease progression.** In 1980 and 1981, experiments were conducted in a field of 65 F6 or F8 lines, respectively, of a Forrest × Semmes cross. Plant heights among these lines varied from 84 to 128 cm. Three-row, 5.5-m-long plots were planted in a randomized complete block design with five replications. Between every two plots (every seventh row throughout the field) a row of Essex soybeans was planted and mechanically inoculated on 17 June 1980 and 15 June 1981 with BPMV to serve as a source of inoculum. Data were collected in both years from each of nine sets of two adjacent rows having plant height differences in each set of at least 10 cm. In 1981, two equally tall (95 cm) and two equally short pairs of adjacent rows were also selected for controls.

At 4, 5, and 6 wk after inoculation of virus source rows in 1980, plant heights were measured at three random positions on adjacent rows of each pair of selected lines, and percent disease incidence and symptom severity ratings were also determined for these rows. Symptom severity and plant height data were also collected from all other lines 6 wk after inoculation. Eighteen samples of symptomatic leaves from rows of each pair were packed in ice, transferred to the laboratory, and subjected to serological tests which confirmed the presence of BPMV in each row. Disease incidence data and plant height measurements were obtained in 1981 as in 1980 except that disease incidence data were collected 5.5 wk after inoculation.

**Plant-to-plant spread of BPMV within rows.** Disease incidence in plots of Ransom and Centennial soybeans planted on 11 May (early) and 25 June (late) 1982 was monitored weekly beginning 16 June and 15 July, respectively. Plots were arranged to promote unidirectional initial spread of viruliferous beetles from virus-infected soybeans (Fig. 1). The number and position of plants in

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each row were recorded on a map and infected plants were recorded on 10, 16, and 27 July. Ordinary runs analysis (2) was used to determine if disease spread within a row was random or nonrandom.

**Yield compensation.** Cultivars Ransom and Centennial were each planted in 1982 in four, six-row plots (6 m long). To prevent

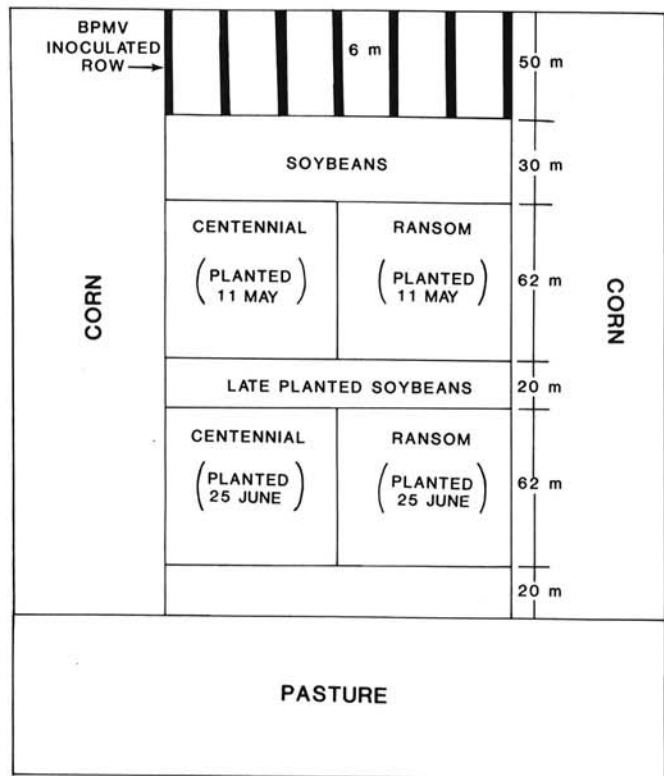


Fig. 1. Field map for location of test plots and virus source plants for monitoring disease progression in early (11 May)- and late (25 June)-planted soybeans in 1982. Soybeans were planted in 43 rows.

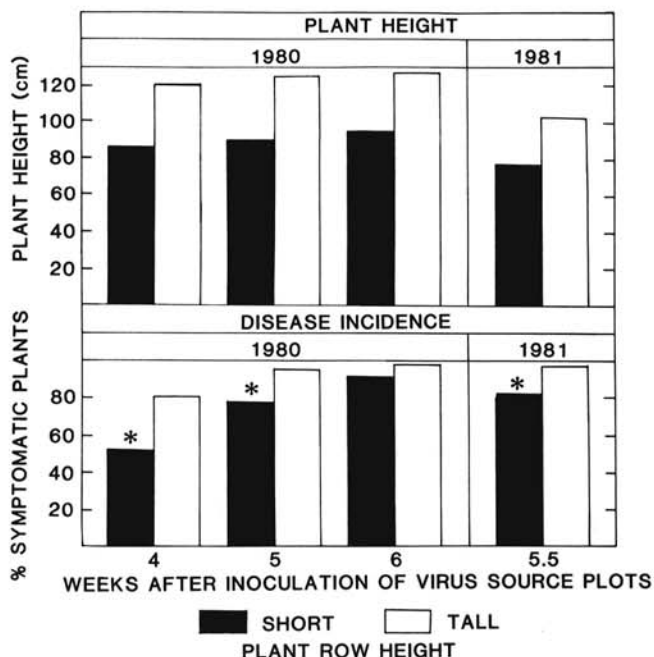


Fig. 2. Average plant height and bean pod mottle disease incidence of adjacent rows of different heights (short and tall) within a field of progeny lines from a Forrest x Semmes cross. Columns of each pair with an asterisk differ significantly according to paired *t*-tests ( $P = 0.05$ ).

natural spread of the virus, the systemic insecticide disulfoton (Di-Syston; Mobay Chem. Corp., Kansas City, MO 64120) was placed in the furrows before planting at a rate of 1.1 kg a.i./ha, and plants were sprayed biweekly with carbaryl (Sevin 80 WP; Union Carbide, Research Triangle Park, NC 27709) throughout the season until pod set. Four rows of soybeans (Ransom) between plots were used as a path for a high-clearance sprayer.

Inoculation treatments were as follows: 1) all plants were mechanically inoculated; 2) all plants were inoculated except for 10 plants, each spaced approximately 1 m apart, which were covered with plastic bags during inoculations; 3) 10 plants, 1 m apart, in

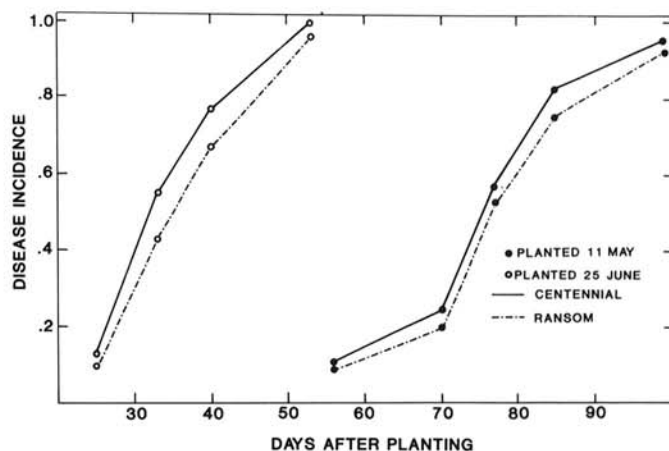


Fig. 3. Progression of BPMV in early- and late-planted soybeans.

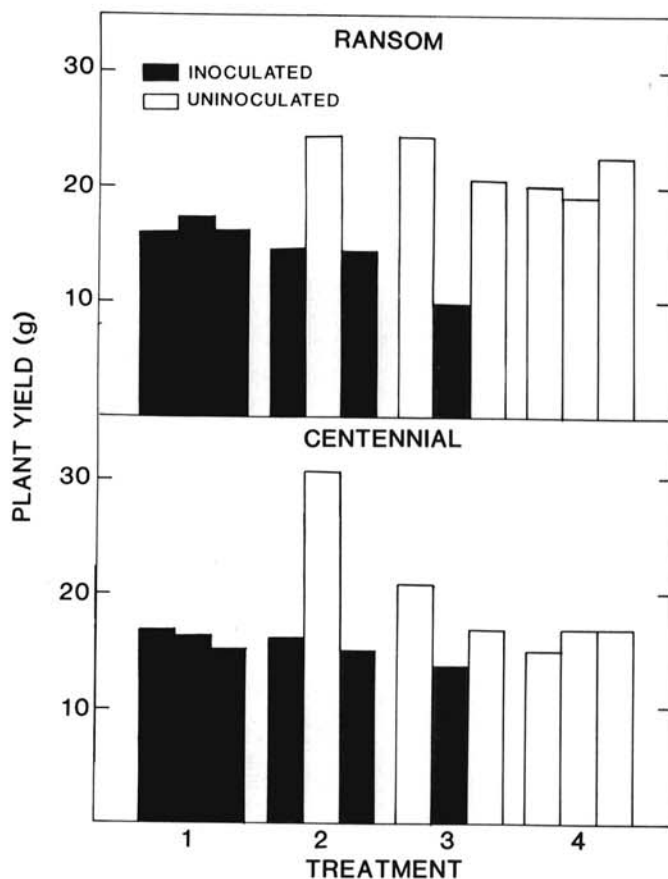


Fig. 4. Effect of BPMV infection on competition among adjacent soybean plants expressed as seed-yield per plant. Plants in treatments 1 and 4 are all inoculated or uninoculated, respectively; center plants in treatment 2 are uninoculated and are adjacent to inoculated plants; center plants in treatment 3 are inoculated and are adjacent to uninoculated plants.

each row were inoculated by rubbing the leaves with a gauze pad soaked in inoculum; or 4) no plants were inoculated. Uninoculated plants in treatment 2 and inoculated plants in treatment 3 were tagged.

On 27 July, 10 sets of triplets (three adjacent plants) were tagged in each of the four middle rows of each plot: sets in which all plants had BPMV symptoms, sets with a symptomless plant between two symptomatic plants, sets with only center plants (tagged at inoculation) symptomatic, and sets with all symptomless plants. On 18 August, triplets were checked to insure that proper disease patterns still existed in each triplet.

## RESULTS

**Plant height effects on disease progression.** In 1980, disease incidence in rows of short plants was less than that in adjacent tall plants on each collection date (Fig. 2). As the season progressed, however, the differences in disease incidence became less. Symptom severity scores were greater for the taller rows than for adjacent rows of shorter plants of each pair, and the correlation between plant height and symptom severity was 0.755. However, when data of plant height and symptom severity were analyzed for all plots in the field without regard to height of neighboring rows, the correlation between plant height (avg 106 cm) and symptom severity (avg 2.3) was only 0.520. In 1981, the disease incidence difference between adjacent rows differing in plant height averaged 13% (Fig. 2). Disease incidence scores of adjacent rows with similar plant heights each averaged 89% and was intermediate to disease incidence scores for tall (97%) and short (83%) rows of adjacent short-tall row pairs.

Tall and short genotypes had essentially equal numbers of nodes in both seasons, but tall genotypes generally had longer internodes and petioles. Leaf blades of tall genotypes appeared to be slightly larger and thus tall plants appeared to be more succulent.

**Planting date effects on disease progression.** To attain 0.1 disease incidence in late planted soybeans required 25 days after planting, whereas this level of disease incidence in the early planted crop required over 55 days (Fig. 3). Approximately half of the late crop was infected after 34 days, but the early planted crop required almost 75 days to reach this level of disease incidence (Fig. 3). Centennial always had greater disease incidence than Ransom, though differences were not significant.

**Plant-to-plant spread within rows.** Virus spread in 1982 was primarily nonrandom in early planted soybeans at 10 wk after planting when disease incidence was 25% or less (Table 1), but as disease incidence increased, virus spread became more random. This nonrandom spread was not apparent in late-planted soybeans.

**Yield compensation.** Uninoculated symptomless plants grown between two virus-infected plants of Ransom and Centennial yielded 14 and 60% more, respectively, than did symptomless plants between two other symptomless plants (Fig. 4). Virus-infected plants grown between uninfected plants of Ransom and Centennial yielded 42.8 and 15.6% less, respectively, than they yielded in 100% infected stands.

## DISCUSSION

The effect of plant height on BPMV disease incidence assumes importance when genotypes of various heights are agronomically evaluated under natural conditions of BPMV dissemination. Earlier virus infection of the taller lines growing beside shorter lines will cause greater yield losses of the tall lines than that of the shorter lines since infection of young plants causes greater yield losses than later infections (7,8). Boiteau et al (1) observed that over 50% of beetles trapped in flight were at levels below the tallest (84 cm) of their soybeans; therefore, plants of taller lines may have blocked beetle flight, and more vectors landed on plants of taller rows than on adjacent rows of short plants. In our study, plants of the taller lines may also have been a more attractive food source since they appeared more succulent than those of the short lines.

Differences in plant height may indirectly affect symptom severity in BPMV-infected soybeans. Symptom severity was found to be inversely correlated with plant age at time of infection (8), and since tall plants adjacent to short plants were infected earlier with BPMV than were the shorter plants, the significant ( $P = 0.05$ ) correlation ( $r = 0.789$ ) between plant height and symptom severity in short-tall adjacent rows could be expected. Since yield loss is also correlated with symptom severity (8), the taller plants probably sustained greater yield reduction caused by the virus than the shorter plants. Windham and Ross (8) also showed that early inoculation of young plants resulted in higher yield loss than did inoculations of plants at later growth stages.

The rapid virus spread in the late-planted soybeans compared to that in early-planted soybeans may have been due to both a larger

TABLE 1. Bean pod mottle disease incidence and number of rows with random or nonrandom virus transmission by beetles in early and late planted soybeans at various dates

Cultivar (planting date)	Rated (day/mo.)	Mean disease incidence	Number of rows with:		
			Nonrandom <sup>a</sup> occurrence	Random <sup>a</sup> occurrence	All healthy (H) or diseased (D) plants
Centennial (11 May)	6 July	0.10	5	2	2 (H)
	20 July	0.25	8	1	0
	27 July	0.56	5	4	0
	4 August	0.83	3	6	0
	18 August	0.95	0	2	7 (D)
Centennial (25 June)	20 July	0.13	5	4	0
	27 July	0.55	3	6	0
	4 August	0.77	3	6	0
	18 August	1.00	0	0	9 (D)
Ransom (11 May)	6 June	0.08	5	2	2 (H)
	20 July	0.20	9	0	0
	27 July	0.63	1	8	0
	4 August	0.78	4	5	0
	18 August	0.93	0	9	0
Ransom (25 June)	20 July	0.10	3	2	4 (H)
	27 July	0.43	2	7	0
	4 August	0.67	5	4	0
	18 August	0.96	2	0	7 (D)

<sup>a</sup> When using ordinary runs analysis, rows with fewer runs of healthy or diseased plants than expected ( $P = 0.05$ ) are considered to be nonrandom (clustered) when disease spread is assumed to be random.

virus reservoir and/or a larger population of vectors with increased flying activity in the late-planted crop. Boiteau et al (1) reported that the first generation of BLB produced from the overwintering population increased in flight activity after a period of intense feeding. This increased vector flight activity probably coincided with the early growth stages of the late-planted soybean crop. Late-planted soybeans grown near early-planted, BPMV-infected soybean fields may sustain considerable yield reduction because of the rapid dissemination of the virus in the young, late-planted soybeans.

An explanation for the early-season nonrandom virus transmission and late-season randomness of virus distribution may reside in the nature of the vector's migratory habits and the frequency of virus source plants. If feeding beetles tended to crawl from plant to plant, after their initial flight from the virus source plant, clumps of infected plants would result. If the initial frequency of viruliferous beetles was relatively low, this would create isolated clusters of infected plants and distribution would be nonrandom. On the other hand, either flights of larger numbers of viruliferous beetles into soybean fields and/or very active flights during feeding of viruliferous beetles would produce a random distribution of infected plants. Soybean plants are often less than 5 cm apart and foliage of adjacent plants intertwine. A foliar feeding, crawling beetle could, therefore, move along a row and fail to feed on certain plants, thus creating skips in runs of infected plants. Since in ordinary runs analysis, a run is any number of either healthy or diseased adjacent plants, these skips would increase the number of runs and hence make the virus distribution appear more random than would be expected if inoculation was done by nonflying vectors.

Healthy soybean plants scattered among BPMV-infected plants may result in yield losses being less than that based on values obtained in studies using 100% infected plants because of yield compensation effects. Since the growth stage at which plants become infected is crucial in determining soybean yield loss, an accurate estimation of number and distribution of plants that escaped early BPMV infection is necessary to accurately compare yield loss data of different genotypes.

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