Effects of Bean Pod Mottle Virus on Soybean Seed Maturation and Seedborne *Phomopsis* spp.

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ABSTRACT

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Maturation rate, measured as length of late-season growth stage intervals, was studied in diverse soybean genotypes to determine if bean pod mottle virus (BPMV) infection altered the role of plant and seed drydown when seed infection by *Phomopsis* spp. increased. Foliage symptoms, stunting, and significant yield reductions due to BPMV occurred in all but one of the 12 soybean entries. Pod infection was consistently increased by BPMV, whereas seed infection was significantly increased in genotypes only if the virus infection delayed the rate of seed maturation. BPMV delayed maturity by extending the length of late-season growth stage intervals and increased seed infection in the soybean entries susceptible or moderately resistant to seed diseases. Effects of BPMV on soybean genotypes that matured under the same environmental conditions indicate that initial levels of seed infection are directly related to the rate of pod and seed maturation. Modifications by BPMV in the R7–R8 interval that delayed harvest maturity of soybean entries susceptible and moderately resistant to seed infection were correlated with increases in levels of seedborne *Phomopsis* spp.

Numerous viruses that infect soybean (*Glycine max* (L.) Merr.) are reported to delay maturity (8). Specific reference to virus infection and reductions in seed quality (increased frequency of moldy seed) are associated with tobacco ringspot virus (TRSV), bean pod mottle virus (BPMV), and specific strains of soybean mosaic virus (SMV) (2,6,8,9). Soybean virus studies by Stuckey et al (9) involving BPMV, SMV, and bean yellow mosaic virus (BYMV) identified an increase in the susceptibility of soybean to seed infection by *Phomopsis* spp. only with BPMV, but the mechanism underlying this interaction was not suggested.

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Seed infection caused by *P. sojae* Lehman and *P. longicolla* T.W. Hobbs is the most serious seed disease of soybean. A rapid rate of seed maturation (moisture loss) during the R7–R8 growth stage interval has recently been identified in soybean genotypes resistant to seed infection (1,5). Susceptible genotypes in the same environments demonstrated a somewhat slower rate of seed maturation. Incidence of seed infection in the susceptible genotypes was also routinely modified by growth regulator treatments that increased or decreased the rate of seed maturation (1).

The effects of BPMV on soybean genotypes have been investigated previously in southern U.S. production environments (3,6,9). However, its effects on *Phomopsis* spp. colonization of both pods and seeds in diverse soybean germ plasm or on rate of seed maturation have received little attention. The objective in this study was to determine if BPMV modifies the rate of pod and seed matur-

ation, and, consequently, if maturation modified by the virus infection alters the development of *Phomopsis* seed infection.

MATERIALS AND METHODS

Field experiments were conducted on the Purdue University Agronomy Farm, Lafayette, Indiana, in continuous soybean plots with conventional tillage using 12 soybean entries representing diverse germ plasm and maturity groups I, II, and IV. The BPMV isolate used came from the Purdue virus collection (R. Lister, *personal communication*) and was similar to the Kentucky isolate (9) that increased incidence of seed infection by *Phomopsis* spp. The virus was increased in soybean cv. Amsoy 71, and inoculum was processed as described by Stuckey et al (9). Plants of each soybean entry were inoculated in June when the majority of the entries were at a V4 or V5 growth stage (four or five nodes, respectively, on the main stem had fully developed leaves, beginning with the unifoliolate nodes). Plants in the two center rows of a plot were inoculated. Two trifoliolate leaves per plant were sprayed at 70 psi with inoculum from an airbrush connected to a tank containing carbon dioxide.

The soybean entries previously identified as resistant and susceptible to fungal seed infection (1,5) were planted on 15 May 1985 and on 23 May 1986. Since environmental conditions during final stages of seed maturation have a dominant influence on fungal infection of seed, resistant and susceptible soybeans that mature in the same environment were compared. Soybean entries resistant to seed infection (PI 404169A, PI 416946, PI 417274, PI 417460, Gnome, and PI 80837) and entries susceptible to seed infection (PI 361093, Miami, Amsoy 71, PI 361065B, PI 417520, and PI 361095) were paired by maturity group (I, II, II, II, II, and IV, respectively). The group II pairs were different in subgroup maturity (e.g., pair 2 was an early group II, whereas pair 5 was a late group II). Soybean entries were planted in a randomized complete block with three replications. Each plot consisted of four rows, each 2 m long and 0.76 m apart. A split-split plot design was used, with paired entries as main plots, seed disease reaction of entries as subplots, and virus treatments as subsubplots. Two virus treatments—a noninoculated control and inoculation with BPMV—were imposed. Selected plants from each treatment were collected early in the growing season and tested by DAS-ELISA to verify infection by BPMV (4). Plants were rated at 40 days after inoculation for virus symptom severity based on a scale from 1 to 5, where 1 = no visible symptoms, 2 = slight stunting and no apparent mottling symptoms on leaves, 3 = moderate stunting and mild mottling symptoms on leaves, 4 = moderate to severe stunting and striking mottling symptoms on leaves, and 5 = severe stunting and mottling symptoms on leaves.

The occurrence of reproductive stages with special emphasis on growth stages R6, $R7_1$, $R7_3$, and R8 as previously described (1,4) was recorded throughout the growing season. The $R7_1$ and $R7_3$ substages, identified by 1 and 50% pods with mature pod color (i.e., brown or black), respectively, are critical to fungal invasion of seed during the R7–R8 drydown interval.

Fungal infection of pods and seed at the above mentioned growth stages was recorded for control and BPMV-inoculation treatments. Pods containing seed were collected randomly from the main stems of 10 different plants in the middle two rows of each plot. Pod and hand-shelled seed samples were separated, surface-disinfested by immersion in 95% ethanol for 20 sec and 1% sodium hypochlorite for 1 min, and then placed on PDA in 9-cm-diameter culture dishes. After incubation for 7 days at 24–26 C, the samples were evaluated for fungal growth. All pod- and seedborne microorganisms were recorded; however, only data on infections by *P. sojae* and *P. longicolla* will be presented.

Data on plant height, seed yield, and seed size were taken for each plot when plants reached full maturity (R8). A harvest (R8) seed sample (mechanically threshed) from the two center rows of each plot also was used for a bulk seed assay to verify the R8 hand-shelled seed sample data. Two 100-seed subsamples were taken from the harvest or yield sample to determine seed size (milligrams per seed). One of these subsamples was also assayed for seedborne *Phomopsis* spp. as described above.

RESULTS

BPMV infections occurred in all soybean entries and altered plant development both years, but more so in 1985 than in 1986. On the basis of early-season DAS-ELISA test data and the rating data for virus symptoms on foliage at 40 days after inoculation, virus infection occurred only in inoculated plants. Plant development (height, yield, seed size) in all plots was reduced by early-season drought conditions in 1986. Plants in all entries except Miami and PI 361095 were shorter in 1986 than in 1985, ranging from 3.8% shorter in PI 361065B to 54.6% shorter in PI 416946. Entries with a determinate growth habit (PI 404169A, PI 361093, PI 416946, PI 417274, PI 417460, Gnome, and PI 80837) showed a more severe reduction in height than indeterminate entries (Miami, Amsoy 71, PI 417520, PI 361065B, and PI 361095). Reductions in seed yield and seed size were similar to the height reductions attributed to the 1986 early-season drought stress. Weather conditions previously described (1) were also more favorable in 1985 than in 1986 for seed infection by *Phomopsis* spp. during seed maturation in the early maturing entries. The 1985 data will be presented to emphasize the effects of BPMV infections on soybean entries resistant and susceptible to *Phomopsis* seed infection.

Data on agronomic characteristics and BPMV effects for the 12 soybean genotypes are presented in Table 1. Virus severity ratings associated with BPMV inoculation averaged 3.2 and ranged from 1.5 in PI 417460 to 4.0 in PI 417520 and PI 361093. The severity rating averaged 2.6 for genotypes resistant to *Phomopsis* seed infection and 3.8 for susceptible genotypes. Plants inoculated with BPMV were shorter in all entries. Height reduction averaged 19% and ranged from 5% in PI 417460 and Gnome to 36% in PI 361095. Correlations between BPMV severity ratings and height reductions due to BPMV were positive ($r = 0.772$) and significant at the $P = 0.01$ level. Yield losses due to BPMV averaged 42% across all entries and were all statistically significant except for PI 417460, which had only a 6% reduction in yield. Average seed size decreased 6%, but the decrease was significant in only four entries—Amsoy 71, PI 417520, PI 80837, and PI 361095. Correlations between BPMV severity ratings and yield reductions due to BPMV were positive ($r = 0.790$) and significant at the $P = 0.01$ level.

Alterations in the length of time between planting and full or harvest maturity (Table 1) were mainly due to changes in duration of late-season growth stage intervals (Table 2). Two intervals, the late seed filling and early drydown interval ($R6–R7_1$) and the major drydown interval, beginning physiological maturity to full maturity ($R7_1–R8$), were used to determine the effect of BPMV on maturation rate. The average number of days to R8 was 127 in *Phomopsis*-resistant entries and 129 in susceptible entries. Infection by BPMV delayed the maturity date an average of 3 days (131 in BPMV-inoculated plants vs. 128 in control plants); four entries (PI 417460, PI 417274, PI 361065B, and PI 80837) showed little or no delay, and the remaining eight entries showed significant delays of 3–7 days. Significant delays in full maturity occurred in two of the entries resistant to seed diseases and five of the susceptible entries. The significant delays in length of time between planting and full maturity were due to reduced rates of maturation during the $R6–R7_1$ and $R7_1–R8$ intervals.

Numerous fungi infected pods and seeds, but species of the *Diaporthe/Phomopsis* complex (6,8), referred to as *Phomopsis* spp., were the most prevalent both years and will be presented using 1985 data. *Cercospora kikuchii* (Matsumoto & Tomoyasu) M.W. Gardner

and *Alternaria* spp. were also more prevalent as pod pathogens than other common soybean pathogens, but they did not cause high levels of seed infection.

The average pod infection by *Phomopsis* spp. in resistant and susceptible entries is presented in Table 3. Pod infection, which has been reported to occur as early as R3, was extensive at the R6 growth stage and significantly higher in plants inoculated with BPMV. Although resistant and susceptible entries differed significantly in *Phomopsis* spp. infection at the R6 growth stage, it was the group of entries resistant to fungal seed infection that had higher pod infection in both control and BPMV-inoculated plants. However, at $R7_1$, when pod infection increased to ≥60% in the control plants and ≥70% in BPMV-inoculated plants, there were no significant differences between resistant and susceptible entries. This indicated that the levels of pod inoculum were similar for plants in the control and BPMV treatments at the time when seed infection began.

The BPMV infection significantly increased *Phomopsis* spp. infection in pods at $R7_3$, but there were still no significant differences in pod infection between the two groups of plants (resistant vs. susceptible to seed diseases). Significant differences occurred in *Phomopsis* spp. infection between resistant and susceptible entries in control and BPMV treatments at the R8 stage. The susceptible entries in the control and BPMV treatments averaged 29 and 46% pod infection, respectively, whereas the resistant entries averaged 36% in each treatment.

The incidence of *Phomopsis* spp. infection of pods declined markedly between growth stages $R7_3$ and R8 (from 59 to 36% in the resistant entries and from 61 to 29% in the susceptible entries for the control treatment, and from 76 to 36% in the resistant entries and from 77 to 46% in the susceptible entries for the BPMV treatment). The percentages of infected pods from resistant and susceptible genotypes (Table 3) were similar during the $R7_3$ growth stage.

With respect to seeds, little or no infection occurred prior to physiological maturity ($R7_1$) in control or BPMV-inoculated plants (Table 3). There was a sharp increase in seed infection of the susceptible entries after $R7_1$. Seed infection in resistant entries at $R7_1$ averaged 1% in control plants and 13% in BPMV-inoculated plants, whereas seed infection in susceptible entries averaged 13% in control plants and 24% in BPMV-inoculated plants. The average seed infection by *Phomopsis* spp. in resistant entries remained constant at the $R7_3$ and R8 growth stages, and the control (5–6%) and BPMV (13–14%) treatments were not significantly different. Average percentages of seed infection in susceptible entries increased to 29 and 26% in the control and to 46 and 42% in the BPMV treatments at the $R7_3$ and R8 growth stages, respectively; these percentages were significantly higher than the mean percentages in the control treatment.

Modifications by BPMV in the R6–R8 interval that delayed harvest maturity of soybean entries susceptible and resistant to fungal seed infection were directly associated with increases in levels of seedborne *Phomopsis* spp. Correlations between seed infection and duration of the interval during which major seed infection occurred ($R7_1$–R8) were positive ($r = 0.646$) and significant at the $P = 0.01$ level; the R6–$R7_1$ interval and the total seed maturation interval (R6–R8) were positive but not significant. Data for duration of the selected growth stages (R6–$R7_1$, $R7_1$–R8, and R6–R8) and percent seed infection by *Phomopsis* spp. at harvest maturity for the individual entries are presented in Table 2. The significant increases in the level of seed infection were always associated with variation in length of either the R6–$R7_1$ or the $R7_1$–R8 interval. Increases in the length of a selected growth stage interval (as a result of BPMV inoculation) were not always significant but were accompanied by increases in the incidence of

Table 1. Effects of bean pod mottle virus on agronomic characteristics of individual soybean entries[w]

Entry Treatment[x]	Virus severity rating[y]	Height (cm)	Yield (gm/4 m)	Seed size (mg/seed)	Harvest maturity (days)
PI 404169A					
Control	1.0	86.0	578	145.3	120
BPMV	3.3	66.3*[z]	398*	142.0	127*
Virus reduction (%)		23	31	2	
PI 361093					
Control	1.0	69.7	1,093	178.0	117
BPMV	4.0	53.0*	353*	171.0	124*
Virus reduction (%)		24	68	4	
PI 416946					
Control	1.0	96.3	709	131.7	121
BPMV	3.2	75.9*	229*	125.7	125*
Virus reduction (%)		21	68	5	
Miami					
Control	1.0	97.3	1,299	197.7	125
BPMV	3.5	75.9*	578*	191.0	128*
Virus reduction (%)		22	56	3	
PI 417274					
Control	1.0	97.3	954	153.7	122
BPMV	1.8	90.3	705*	144.0	123
Virus reduction (%)		7	26	6	
Amsoy 71					
Control	1.0	102.0	1,213	197.0	129
BPMV	3.7	75.4*	380*	173.3*	134*
Virus reduction (%)		26	69	12	
PI 417460					
Control	1.0	69.0	978	226.0	130
BPMV	1.5	65.7	922	216.7	131
Virus reduction (%)		5	6	4	
PI 417520					
Control	1.0	90.7	850	178.3	128
BPMV	4.0	67.7*	466*	166.7*	133*
Virus reduction (%)		25	45	6	
Gnome					
Control	1.0	69.7	1,564	180.7	131
BPMV	3.3	66.4	866*	177.0	135*
Virus reduction (%)		5	45	2	
PI 361065B					
Control	1.0	89.7	939	176.0	133
BPMV	3.5	74.1*	590*	169.7	133
Virus reduction (%)		17	37	4	
PI 80837					
Control	1.0	79.7	1,142	186.0	141
BPMV	2.7	68.5*	804*	167.3*	142
Virus reduction (%)		14	30	10	
PI 361095					
Control	1.0	131.0	1,185	156.3	143
BPMV	3.8	84.4*	502*	137.0*	147*
Virus reduction (%)		36	58	12	

[w] Data are means of three replications in 1985.

[x] Control = not inoculated; BPMV = inoculated with bean pod mottle virus at the V4–V5 growth stage.

[y] Rating based on a scale of 1–5, where 1 = no visible symptoms and 5 = severe stunting and mottling symptoms on leaves.

[z] * = BPMV mean is significantly different from control mean based on Waller-Duncan-Bayesian LSD test (k-ratio = 100).

Table 2. Time intervals between growth stages associated with soybean seed maturation (R6–R8) and seedborne *Phomopsis* spp. at full maturity (R8)[w]

Entry Treatment[x]	Maturity group	Interval[y] days			*Phomopsis* seed infection (%)
		R6–$R7_1$	$R7_1$–R8	R6–R8	
PI 404169A	I				
Control		12	12	24	11.7
BPMV		17*[z]	13	30*	23.7*
PI 361093	I				
Control		16	11	27	37.0
BPMV		18*	14*	32*	51.3*
PI 416946	II				
Control		13	11	24	19.3
BPMV		15	11	26	35.7*
Miami	II				
Control		23	12	35	28.0
BPMV		24	13	37	51.3*
PI 417274	II				
Control		19	9	28	1.3
BPMV		19	8	27	2.0
Amsoy 71	II				
Control		26	13	39	27.0
BPMV		26	14	40	50.0*
PI 417460	II				
Control		26	9	35	4.0
BPMV		26	8	34	3.7
PI 417520	II				
Control		21	13	34	18.3
BPMV		24*	13	37*	37.7*
Gnome	II				
Control		26	10	37	1.0
BPMV		25	13*	38	3.0
PI 361065B	II				
Control		19	19	38	9.7
BPMV		18	19	37	13.3
PI 80837	IV				
Control		19	11	30	3.0
BPMV		19	11	30	5.3
PI 361095	IV				
Control		23	11	34	25.7
BPMV		25	13	38*	39.3*

[w] Data are means of three replications in 1985.
[x] Control = not inoculated; BPMV = inoculated with bean pod mottle virus at the V4–V5 growth stage.
[y] R6–$R7_1$ = late seed filling interval; R6 = full green bean seed stage to $R7_1$ = beginning of physiological maturity (1% of pods with mature color); $R7_1$–R8 = major drydown interval, beginning of physiological maturity to full or harvest maturity (95% of pods with mature color); R6–R8 = late seed filling and maturation interval.
[z] * = BPMV mean is significantly different from control mean based on Waller-Duncan-Bayesian LSD test (*k*-ratio = 100).

Table 3. Effects of bean pod mottle virus on pod and seed infection by *Phomopsis* spp. at different growth stages in six pairs of soybeans entries

		Percent *Phomopsis* infection[x]							
		R6		$R7_1$		$R7_3$		R8	
Treatment[v]	Entries[w]	Pod	Seed	Pod	Seed	Pod	Seed	Pod	Seed
Control	Resistant	29 a[y]	00 a	64 a	01 a	59 a	06 a	36 b	05 a
	Susceptible	14 b	00 a	62 a	13 b	61 a	29 b	29 a	26 b
BPMV	Resistant	36 a*[z]	00 a	72 a	13 a*	76 a*	14 a	36 a	13 a
	Susceptible	27 b*	00 a	71 a	24 b*	77 a*	46 b*	46 b	42 b*

[v] Control = not inoculated; BPMV = inoculated with bean pod mottle virus at the V4–V5 growth stage.
[w] Seed infection reaction to *Phomopsis* spp. Resistant = soybean entries PI 404169A, PI 416946, PI 417274, PI 417460, Gnome, and PI 80837; susceptible = soybean entries PI 361093, Miami, Amsoy 71, PI 417520, PI 361065B, and PI 361095.
[x] R6 = full green bean seed stage; $R7_1$ = beginning of physiological maturity (1% of pods with mature color); $R7_3$ = intermediate maturity (50% of pods with mature color); R8 = full or harvest maturity (95% of pods with mature color).
[y] Means followed by the same letter within a column and treatment are not significantly different (*k*-ratio = 100) according to Waller-Duncan-Bayesian LSD test.
[z] * = BPMV treatment × entry mean is significantly different from the respective control treatment × entry mean based on Waller-Duncan-Bayesian LSD test (*k*-ratio = 100).

seedborne *Phomopsis* spp. except in Gnome. The R6–$R7_1$ period appeared to have an effect on seed infection for four of the susceptible genotypes (PI 361093, Miami, PI 417520, and PI 361095) and for two of the resistant genotypes (PI 404169A and PI 416946). This R6–$R7_1$ delay in maturation was significant only in PI 404169A, PI 361093, and PI 417520. The $R7_1$–R8 period also appeared to have an effect on seed infection for PI 404169A, PI 361093, Miami, Amsoy 71, and PI 361095. The $R7_1$–R8 delay in maturation was significant for both PI 361093 and Gnome, but a significant increase in seed infection by *Phomopsis* spp. did not occur in Gnome.

All susceptible entries, with the exception of PI 361065B, showed a delay in seed maturation and a significant increase of seedborne *Phomopsis* spp. when inoculated with BPMV. The BPMV inoculation did not cause a significant increase in *Phomopsis* spp. in the four entries with the highest levels of seed disease resistance (PI 417274, PI 417460, Gnome, and PI 80837). With the exception of Gnome, maturation rates during the R6–$R7_1$ or $R7_1$–R8 interval for these highly resistant entries were not delayed by BPMV infection. The reactions of the other two resistant entries (PI 404169A and PI 416946), with lower levels of resistance, were similar to those of the susceptible entries, and significant increases in seed infection by *Phomopsis* spp. occurred during the extended drydown period.

DISCUSSION

Host factors as well as BPMV infections that modify late-season seed maturation can influence seed infection by *Phomopsis* spp. (Tables 2 and 3), as evidenced by the results of this study. Modifications in late-season maturation rate by BPMV and their effects on soybean seed infection have not been reported by other researchers. The mechanism underlying the predisposition of BPMV-infected plants to seed infection appears to be related to the maturation rate. But not all soybean entries were predisposed to *Phomopsis* seed infection by the BPMV inoculation. Soybean entries with the highest levels of resistance to seed diseases (PI 417274, PI 417460, Gnome, and PI 80837) had low levels of seedborne *Phomopsis* spp. in both the control and BPMV treatments. This may indicate that seed disease resistance in these genotypes is more stable than in the other two resistant genotypes (PI 404169A and PI 416946). It may also indicate that the genotypes vary in their resistance to BPMV. Evidence of BPMV infection in all entries was initially verified by the ELISA tests, but the virus severity ratings and agronomic characteristics (i.e., height and yield reductions) in the BPMV treatment confirmed that the genotypes vary in resistance to

BPMV (Table 1).

The pairing of soybean entries with similar maturity dates but resistant or susceptible to seed infection was important because it permitted the comparison of BPMV effects on maturation rate and seed infection by *Phomopsis* spp. between genotypes that matured under similar environmental conditions. Maturation rate and duration of growth stage intervals were modified by BPMV (Table 2). Infection by BPMV delayed maturity by increasing late-season growth stage intervals, but these changes were not significant in some of the resistant entries. The delays in maturity by BPMV in this study were similar to delays induced by the growth regulator chloroflurenol (1). The delay in maturity in BPMV-infected plants was somewhat similar to the greenstem syndrome (7), in which stems of BPMV-infected soybeans remained green after pods matured and leaf petioles, but not blades, were still attached. The difference between the two situations is that in this study, BPMV also delayed leaf and pod maturity. This resulted in plants remaining green for a longer time and, consequently, in a slower drydown for pods and seeds. Since major seed infection occurs between physiological maturity and full maturity ($R7_1$–R8), the duration of this interval influences the frequency of seedborne fungi. Seed disease development is enhanced by warm, moist conditions at the time of maturation, and environmental factors that delay or reverse pod drying increase seed infection (1,7,8). Thus, the longer the period in which seed infection can take place, the higher the potential for increased levels of infection. The results obtained with BPMV inoculations support this concept because higher levels of seedborne *Phomopsis* spp. occurred when the R6–$R7_1$ or $R7_1$–R8 interval was increased (Table 2).

This study presents evidence that BPMV increases pod infection by *Phomopsis* spp. (Table 3), but this increase in pod infection did not alter the time of seed infection. Although pod infection was high, few, if any, seeds were infected prior to physiological maturity. If only susceptible soybean entries are considered, the BPMV increases in seedborne *Phomopsis* spp. would appear to be due to the increases in pod infection. However, the pod infection data for resistant soybean entries in both the control and BPMV treatments at full maturity (R8) illustrate that seed infection is much lower than pod infection.

A reduction in plant height was observed in BPMV-inoculated plants when compared with control plants (Table 1). However, entries with the highest levels of resistance to seed diseases (PI 417274, PI 417460, Gnome, and PI 80837) showed less height reduction due to BPMV and had lower BPMV foliage severity ratings than the susceptible entries to which they were paired for maturity (Amsoy 71, PI 417520, PI 361065B, and PI 361095, respectively). Except for Gnome, maturity was not delayed in any of these highly resistant entries, and the duration of the $R7_1$–R8 interval was not increased due to the virus inoculation. The length of the R6–$R7_1$ period was not changed either, even for Gnome. The inability to modify the maturation rate in these highly resistant entries may account for the similar levels of seedborne *Phomopsis* spp. found in BPMV-inoculated and control plants (Tables 2 and 3). Although it appeared that entries with resistance to seed diseases were not affected by BPMV inoculations, significant yield reductions were observed in all but one (PI 417460) of the entries (Table 1). Susceptible and moderately resistant genotypes also suffered significant yield losses consistent with those reported for soybean in southern U.S. production environments (3,6,9). Across all entries, BPMV ratings were statistically significant and positively correlated with height and yield reductions.

The effect of BPMV on soybean maturity has been attributed to an inhibition of pod formation (up to 40%) and to increased pod abortion and shedding that result in plants remaining green after normal plants have matured (8,10). The delay in drydown in plants infected with BPMV, as observed in this study, is a major factor indicating why BPMV-infected plants have higher frequencies of seedborne fungi. Slower drydown at the critical time when pathogens begin seed colonization is one explanation for this phenomenon.

The evaluations within the $R7_1$–R8 interval (Table 3) indicated that major seed infection occurred in both control and BPMV treatments during the $R7_3$–R8 substage interval when pod and seed moisture values decline. These results agree with previous reports regarding time of major seed infection (1,7). Comparison of the 12 diverse germ plasm entries with and without BPMV inoculation indicates that BPMV infection extends drydown periods, allowing increased levels of seedborne *Phomopsis* spp. (Tables 1–3). Our results do not rule out other factors that could determine or influence the levels of seed infection. In fact, other resistance mechanisms may be modified when soybean plants are infected with BPMV. But the research presented here involving BPMV effects on late-season maturation rate presents substantial evidence that when BPMV infections delay the rate at which soybean plants lose moisture during the final stages of seed maturation, seed infection by *Phomopsis* spp. increases.

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