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Bean pod mottle virus: A Threat to U.S. Soybean Production

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Bean pod mottle virus (BPMV) is widespread in the major soybean-growing areas in the southern and southeastern United States. A severe outbreak of BPMV in the north central and northern Great Plains states is currently causing serious concern to soybean growers and to the soybean industry in this region (30). BPMV is efficiently transmitted in nature, within and between soybean fields, by several species of leaf-feeding beetles. The deleterious effects of BPMV infection not only reduce yield but also reduce seed quality, as seeds from infected plants may be discolored. Furthermore, BPMV predisposes soybeans to *Phomopsis* spp. seed infection (85), a major cause of poor seed quality in soybean (78). The recent BPMV outbreak is linked to the warm winters of the past few years that have allowed the beetle vectors to overwinter and emerge in the spring in unprecedented numbers (Fig. 1).

Virion Properties and Genome Organization

BPMV is a member of the genus *Comovirus* in the family *Comoviridae* (93). Like other comoviruses, BPMV has a bipartite positive-strand RNA genome consisting of RNA-1 and RNA-2, which are separately encapsidated in isometric particles 28 nm in diameter (Fig. 2). Virions can be separated by density gradient centrifugation into three components designated top (T), middle (M), and bottom (B). The T component contains empty particles, whereas the M and B components contain single molecules of RNA-2 (approximately 3.6 kb) or RNA-1 (approximately 6.0 kb), respectively. The three components have identical protein composition, consisting of 60 copies each of a large (L) and small (S) coat protein (CP) of 41 kDa and 22 kDa, respectively. The S-CP occurs in two major size classes, the intact protein and a C-terminus truncated version. As a consequence of this heterogeneity, BPMV virions have two electrophoretic forms, a slow- and a fast-migrating form, each containing both M and B nucleoprotein components. Intact S-CP converts to the C-terminus-truncated form with ageing of the virions and involves a specific, yet little understood, proteolytic processing at the C-terminus (47).

BPMV genomic RNAs are polyadenylated, and each has a small basic protein, VPg, covalently linked to its 5′ terminus. The BPMV genome is expressed via the synthesis and subsequent cleavage of large polyprotein precursors (47). The complete nucleotide sequences of the two genomic RNAs of BPMV strain KY-G7 have been reported (13,49). BPMV RNA-1 encodes five mature proteins required for replication (from 5′ to 3′: a protease cofactor [32K], a putative helicase [58K], a viral genome-linked protein [VPg], a protease [24K], and a putative RNA-dependent RNA polymerase, RdRp [87K]), whereas RNA-2 encodes a putative cell-to-cell movement protein and the two coat proteins (13,49).

Historical Perspective

Zaumeyer and Thomas first described BPMV in 1948 on *Phaseolus vulgaris* L. var. Tendergreen. In 1948, the virus was noted to be readily transmitted mechanically, and the experimental host range included several varieties of all groups of snap and dry beans. In further exploration of the BPMV experimental host range, 25 species including 20 genera of plants were evaluated for susceptibility. In this test,

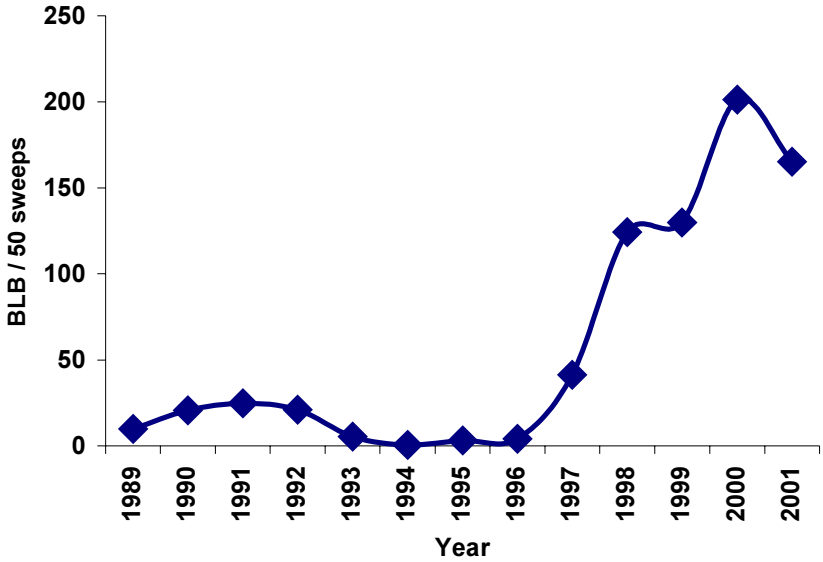

Fig. 1. Number of F_2 bean leaf beetles per 50 sweeps from 1989 to 2001 in central Iowa. Means determined by pooling data from a weekly sampling program conducted on three fields at the Iowa State University Johnson Farm, Ames. Courtesy of Larry P. Pedigo, Wai-Ki F. Lam, and Rayda K. Krell, Entomology Department, ISU.

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some varieties of lima bean (*Phaseolus lunatus* L.) and soybean (*Glycine max* L.) were determined to also be susceptible (98). BPMV was first identified as a soybean problem in the field in 1951 in Arkansas (87). In 1958, the experimental host range list was expanded to include *Lespedeza* sp., *Stizolobium deeringianum* Bort., and *Trifolium incarnatum* L. (82).

Between the 1960s and the 1980s, most BPMV research involved soybean response and studies on inoculation timing relative to plant development and its impact on yield (34,55,65,70,79,96). Studies which established the bean leaf beetle (BLB) (*Cerotoma trifurcata* Forster) (Fig. 3) as the primary vector of BPMV were performed in the 1960s and established the BLB as the most important vector in southern states (60,68,88). Until recently, BPMV research was confined to the southern United States, including the Carolinas, Kentucky, Mississippi, and Arkansas. With the recent movement of this virus into the north central region of the United States, new interest in the pathology of BPMV has arisen.

Distribution of BPMV

After the initial discovery of BPMV in soybeans in Arkansas (87), other states confirmed its presence. BPMV was confirmed in North Carolina and Virginia (82), Kentucky (27), Mississippi (62), and Louisiana (35). In the north central region, BPMV has been confirmed in Iowa (64), Illinois (51), Indiana (K. Perry, *personal communication*), Kansas (29), Nebraska (45), Ohio (17), South Dakota (43), and Wisconsin (44) and has also been reported in Canada (50). BPMV is likely present in all soybean-producing states, but documentation is incomplete.

BPMV is the most common viral pathogen of soybean in several states. In Kentucky, BPMV was found in 66% of 382 fields in 1985 to 1987; incidence varied from low to high within fields (26). Viral incidence was highest in the last year (1987) of the Kentucky survey. In North Carolina, 56 fields were surveyed for viral incidence (73). In the mideastern region of North Carolina, 37% of the fields had more than 50% of the plants infected in 1983. More recently, BPMV incidence has increased significantly in the north central region. For example, 70% of 197 fields sampled in Nebraska had BPMV in 2000 (101). In a recent survey, 73 of 80 counties in Iowa had BPMV (67).

Impact of BPMV Infection on Soybean

Foliage and pod symptoms. Soybean response to BPMV infection varies. Plant symptoms range from a mild chlorotic mottling of foliage to a severe mosaic, with the most obvious symptoms appearing on younger leaves (Fig. 4A, C, D) (69,89). Depending on the soybean variety, BPMV may cause terminal necrosis and death (79). BPMV delays maturity of soybean stems, causing "green stem" (Fig. 4B) (79). The pod mottling symptom that is prominent in snap beans is not prominent in many soybean cultivars due to pubescence, but appears in some (Fig. 4E).

Yield reduction. BPMV infection can reduce soybean yield. According to the *Compendium of Soybean Diseases,* yield loss ranges between 3 and 52% (24). Over a broad geographic range, yield reductions between 10 and 40% have been reported (10,36,55,69,85). Impact of BPMV on yield depends upon the time of virus infection relative to plant development (Fig. 5), with early infection giving the highest yield reduction (25). Ross (69) showed that mixed infection with BPMV and *Soybean mosaic virus* (SMV) reduced yield up to 85%. In Louisiana, it was determined that the BPMV infection level needs to be between 20 and 40% of the plant population to cause economic loss (36). Research performed in North Carolina showed that infection of the plants needs to occur before the V6 growth stage to significantly affect yield (71).

Seed coat mottling. Soybeans infected with BPMV may produce seed with mottled seed coats. The mottling originates at the hilum and is also referred to as "bleeding hilum" since hilum color appears to bleed from its normal zone. The coloration of the hilum is the color of the mottling on the seed (Fig. 4F). Quiniones et al. (65) analyzed seed mottling levels as affected by potential synergistic reactions between SMV and BPMV. SMV-infected plants had 92% of the seed lot mottled, and the SMV and BPMV combination had 96% of the seed lot mottled. Soybean varieties differ in the degree of seed mottling in response to BPMV infection (33,100). Mottling of the seed coat is not a reliable predictor of seed coat infection by BPMV.

BPMV has both primary and secondary effects on seed quality. The delay of maturity of the soybean plant and/or the stress of the systemic virus infection has been shown to have secondary effects on the plant. *Phomopsis* seed infection tends to be higher in BPMV-infected soybean plants (1,85). SMV infection also increases *Phomopsis* seed infection (40). Seed infection by *Phomopsis* occurs during the R7 and R8 growth stages, when pod and seed moisture decline. BPMV infection has been shown to extend dry down periods, resulting in increased levels of *Phomopsis* seed infection (1).

BPMV Diversity and Synergism with SMV

The recent BPMV outbreak has prompted researchers in the major U.S. soybean production regions to undertake a

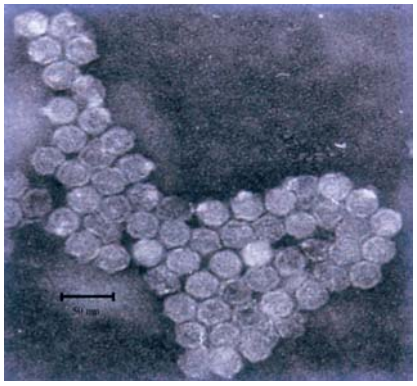

Fig. 2. Negatively stained purified *Bean pod mottle virus* virions (28 nm diameter). Bar equals 50 nm.

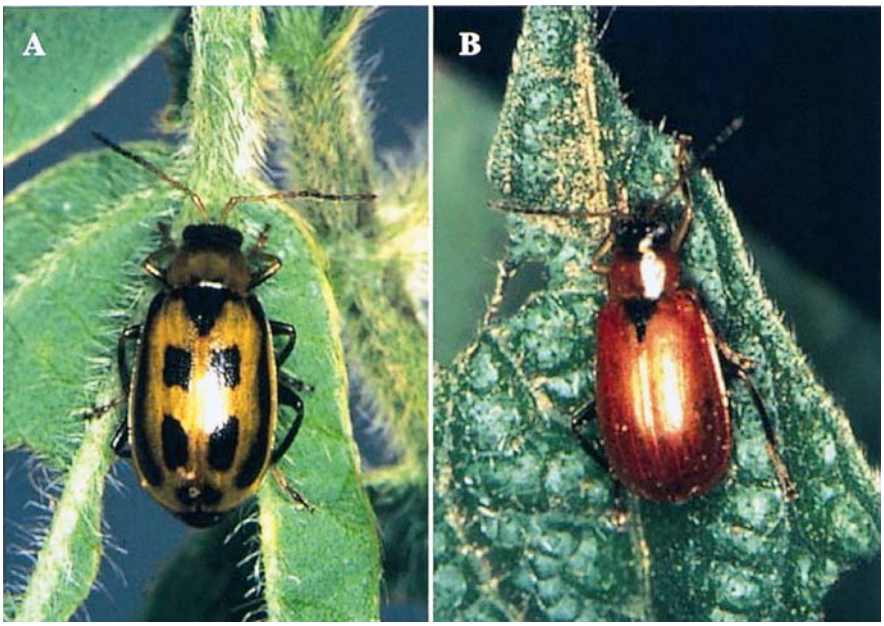

Fig. 3. Bean leaf beetle. A, Common type. B, Red variant. The black triangle behind the thorax is used to distinguish the bean leaf beetle.

concerted effort to screen available soybean germ plasm for resistance/tolerance to BPMV. Until recently, there was no evidence that BPMV existed as multiple strains. Such evidence of genetic diversity is very useful as breeders and others work to develop germ plasm and cultivars that offer broad protection against the full range of BPMV strains as they become known.

The recent work of Gu et al. (30) has revealed at least two genetically distinct BPMV subgroups, I and II. The two subgroups can be clearly distinguished by nucleic acid hybridization analysis (Fig. 6). Furthermore, Gu et al. (30) isolated and characterized naturally occurring reassortants between the two subgroups. It is of interest that isolation of BPMV reassortants coincided with the recent increase of both virus incidence and BLB populations. Under such conditions, one predicts an increased incidence of mixed infections and the emergence of reassortants and sequence variants, the latter possibly originating by RNA recombination events.

BPMV interacts synergistically with the potyvirus SMV, causing drastic reductions in yield and seed quality (3,10,69,85). It is thus prudent to use SMV-resistant cultivars in regions where BPMV is endemic (Table 1). The concentration of BPMV in soybean plants doubly infected with BPMV and SMV is significantly higher (two- to seven-fold, depending on leaf position, i.e., age of infection) than in singly infected plants. SMV titer, however, is not affected by double infection. Enhancement by SMV of BPMV titer in doubly infected plants can be demonstrated in both greenhouse and field-grown plants and is independent of the timing, sequence, or means of inoculation with the two viruses (3,10). Electron microscopic examination of thin sections from doubly infected plants reveals single plant cells containing both SMV and BPMV (3).

Although the mechanism of synergism between SMV and BPMV is not understood, recent studies with synergistic interactions between other pairs of unrelated viruses (in which one of the pair is a potyvirus) suggest that expression of the potyvirus HC-Pro (helper component–protease) gene might interfere with a general antiviral system in plants. Posttranscriptional gene silencing is a candidate for such a host defense system. This in turn allows the nonpotyvirus member of the pair to accumulate beyond its normal level. The HC-Pro thus suppresses gene silencing (2). There appears to be specificity in the interaction between BPMV and SMV since no synergism was detected in BPMV interactions with some other potyviruses, e.g., neither *Bean yellow mosaic virus* nor *Peanut mottle virus* (3). As the mechanism by which HC-Pro suppresses silencing is not known, the answers for why the HC-Pro from SMV, but not from other potyviruses, suppresses gene silencing cannot be addressed as this time.

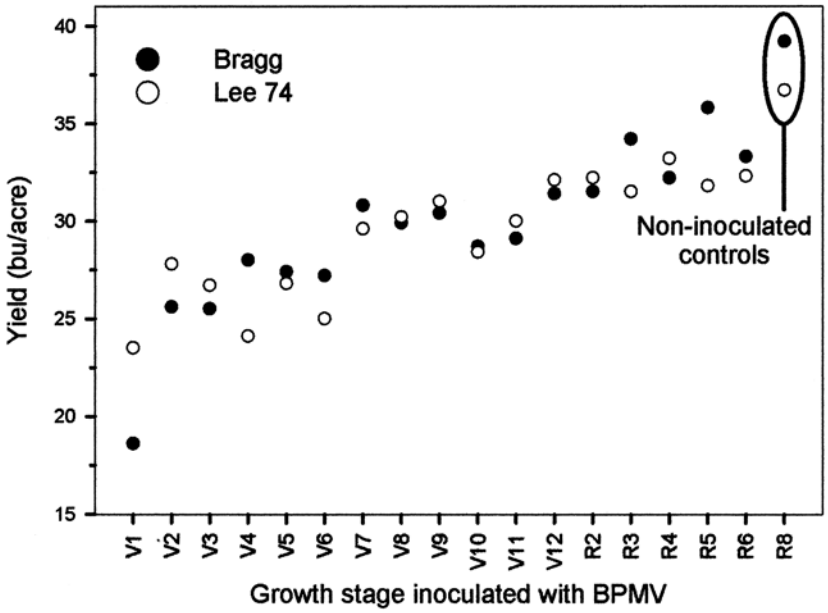

Fig. 5. Impact of *Bean pod mottle virus* (BPMV) on yield of soybean cultivars Bragg and Lee 74, as affected by time of inoculation relative to soybean development. Modified from Hopkins and Mueller (34).

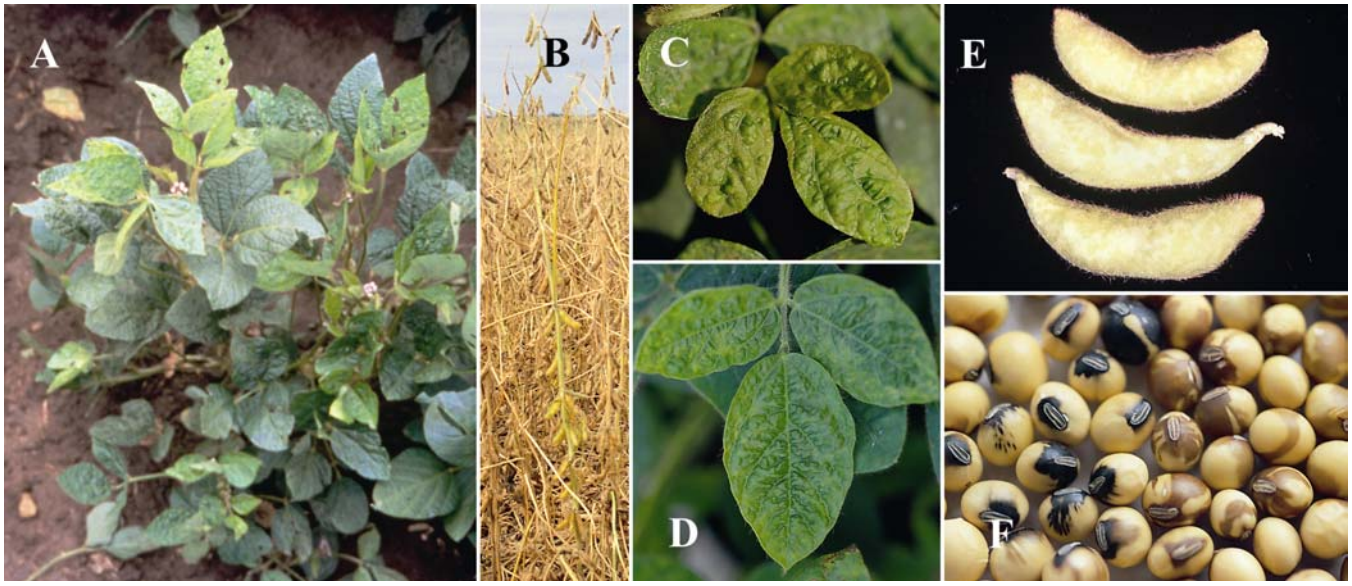

Fig. 4. Symptoms exhibited by soybean plants infected with *Bean pod mottle virus.* A, Plant showing symptoms in younger leaves. B, Green stem symptom in mature field. C and D, Leaves exhibiting rugosity and mottled appearance. E, Pod mottling on cv. Essex. F, Seed coat mottling or bleeding hilum symptom.

Insect Vectors of BPMV

Several leaf-feeding beetles (Coleoptera) in the families Chrysomelidae, Coccinellidae, and Meloidae can transmit BPMV. These include *Cerotoma trifurcata* (Förster) (bean leaf beetle), *Colaspis brunnea* (Fabricius) (grape colaspis), *Colaspis lata* Schaeffer, *Diabrotica balteata* LeConte (banded cucumber beetle), *D. undecimpunctata howardi* Barber (spotted cucumber beetle), *Epicauta vittata* (Fabricius) (striped blister beetle), and *Epilachna varivestis* Mulsant (Mexican bean beetle) (24). More recently, *Diabrotica virgifera virgifera* LeConte (western corn rootworm) and *Odontota horni* Smith (soybean leafminer) have been identified as potential vectors (94). However, in the north central states, the BLB is by virtue of its prevalence the primary BPMV vector (34,60).

Adult BLB overwinter in various habitats, preferring leaf litter in wooded areas in Iowa (41). Temperatures below the critical range of –5 and –10°C cause significant mortality; however, daily leaf litter temperatures in woodlands seldom go below –5°C (42). Ambient air temperatures correlate well with leaf litter temperature above –10°C. Below –10°C, there is a low correlation, and daily leaf litter temperature seldom drops below –2°C. The buffered microhabitat facilitates beetle survival. It is likely that the BLB behaves similarly throughout the north central states, as other work suggests that the beetle overwinters in trash and grass (38), under rocks (20), and in leaf litter (7,54).

In the Midwest, BLB emerge from overwintering sites in mid to late April (83) and move to early legumes, such as *Desmodium* sp. (11) and alfalfa, *Medicago sativa* L. (83,86). Beetles begin to colonize soybeans as seedlings emerge. Viruliferous beetles can potentially transmit BPMV to soybeans at early growth stages, which maximizes reduction in yield and seed quality (70). Phenological studies show the BLB develops one generation per year in much of Minnesota (48), two generations per year in Iowa (83), Illinois (39), and Nebraska (97), and three generations per year in Arkansas and South Carolina (20,38).

The BLB is an extremely efficient virus vector. The beetle may acquire the virus after a single bite, and transmission efficiency increases with time on the virus source plant as well as on the healthy soybean plant (61). Transmission efficiency is found to be very high in F_1 and F_2 beetle populations with 70 to 80% of the beetles that are allowed 72 h acquisition feeding on source plants being able to transmit the virus (S. A. Ghabrial, *unpublished data*). There is no latent period in the vector (23). The virus is not detected in the hemocoel, and transmission is therefore not circulative. The virus is presumably restricted to the digestive tract (92). Retention time is considerable since the virus can be detected in overwintered adults, although virus transmission by these adults may be relatively infrequent. It is probable the virus does not replicate in the beetle since virus level decreases gradually during test feeding on healthy plants (28). Larvae of several chrysomelid beetles transmit beetle-vectored viruses (23), but it is unknown if the larval stages of the BLB can transmit BPMV. Similarly, potential for transovarial transmission of BPMV is unknown. Other beetle-transmitted viruses are not known to be transmitted transovarially.

The spread of BPMV on a local (within and between fields) and a regional basis likely reflects BLB dispersal. Three periods of flight activity were identified in North Carolina, a region with two BLB generations: a field colonization flight followed by trivial and overwintering flights by the first (F_1) and second (F_2) generations (6). The colonizing population exhibited the greatest flight activity, followed by the fall migration to overwintering habitat. Most trivial flights were observed to be ≤30 m.

Inoculum Source for Disease Development

Virus diseases cause the most damage when infection occurs in early stages of soybean growth (Fig. 5). Therefore, elimination of primary sources of BPMV inoculum will facilitate disease management. This is particularly so in view of the lack of commercial cultivars with BPMV resistance. Perennial weeds, seedlings emerging from infected seeds, and overwintering BLB are potential sources of BPMV early in the growing season (45,53,90,91). Some of these reports, however, are preliminary. Roles of overwintering beetles, seed transmission, and weed reservoirs (alternate hosts) in BPMV epidemiology have yet to be critically assessed. The potential for disease control through management of the principal virus vector, the BLB, will be enhanced by accurate identification of the virus source(s).

Alternate hosts. The agriculturally significant hosts of BPMV include soybeans and some *Phaseolus* spp. and cowpea cultivars (24). In the north central United States, these are not believed to act as sources of early inoculum foci from which potentially viruliferous BLB move to emerging soybeans. Alternatively, other leguminous hosts may provide a means for the virus to overwinter and could serve as virus reservoirs. In Iowa, *Desmodium* spp. has recently been demonstrated to be naturally infected with BPMV (R. K. Krell and J. H. Hill, *unpublished*). Although this is the first report in the north central states, Pitre (60) suggested that in Mississippi beetles may become viruliferous after feeding on *Desmodium paniculatum* (L.) DC. Further exploration of potential virus reservoirs is necessary in the north central soybean growing areas. To be a significant

Subgroup I RNA 1	Subgroup I RNA 2	Subgroup II RNA 1	Subgroup II RNA 2	Symptom
✚	✚			Moderate
		✚	✚	Mild
✚			✚	Severe
	✚	✚		Severe

Fig. 6. *Bean pod mottle virus* diversity based on identification of two genetically distinct strain subgroups (I and II) and the occurrence of reassortants. Modified from Gu et al. (30). Symbols in table identify which RNA is present, with yellow and green representing subgroup I and II strains, respectively.

Table 1. Effect of combinations of *Soybean mosaic virus* (SMV) and *Bean pod mottle virus* (BPMV) on soybean yield (modified from Calvert and Ghabrial [10])

	Yield (g/hill) for soybean cultivars		
Virus treatment[x]	Williams	Essex	York[y]
Control	228.2 a[z]	236.8 a	251.4 a
SMV	183.8 b	145.1 b	249.0 a
BPMV	147.6 c	98.6 c	134.7 b
SMV-BPMV	56.1 d	57.6 d	168.4 b
BPMV-SMV	70.8 d	57.2 d	151.2 b

[x] The primary leaves of all test plants, except controls, were inoculated with SMV or BPMV 20 days after planting; in the case of double inoculation treatments, the second virus was applied to the first trifoliolate leaves 1 week later.

[y] The soybean cv. York is resistant to SMV strain G-2, used for inoculation.

[z] Values are means for nine replications. For each cultivar, means followed by the same letter are not significantly different ($\alpha = 0.05$) according to Duncan's multiple range test. Hill size was a 45-cm row containing 20 seeds planted.

virus reservoir in the north central states, a species must serve both as an overwintering host for the virus and as a source for beetle feeding.

Seed transmission. Grower concerns often center on the risk of planting mottled seed and concern that mottled seed will introduce virus. In analysis of this issue, it is important to distinguish between seedborne and seed-transmitted virus. Frequently, seed testing laboratories prepare a seed extract by grinding a group of seeds and detect virus by enzyme-linked immunosorbent assay (ELISA). This detects seedborne virus; detection of seed-transmitted virus is equivocal. Determination of seed-transmission requires grow-out tests where soybean seedlings are tested for virus. It should be noted here that seed transmission generally requires embryo infection, not infection of other seed parts. However, transmission of non-embryo-borne virus is possible as exemplified by the transmission of *Tobacco mosaic virus* (TMV) in tomato and pepper seed (8). TMV is restricted to seed coats, and infection probably occurs through wounds in seedlings contaminated with virus from seed coats.

Numerous efforts have failed to demonstrate seed transmission of BPMV in soybeans. However, there are now two reports of low level (<0.1%) seed transmission of BPMV (45,71). In more recent work with field-harvested seed from two private commercial cultivars, no seed transmission was observed in one cultivar, but 0.037% transmission was obtained in the other (J. H. Hill, *unpublished*). BPMV is stable, easily transmitted mechanically, and is present at relatively high levels in seed coats from BPMV-infected plants (S. A. Ghabrial, *unpublished*). The very low levels of transmission could reflect injury of seedlings contaminated with virus from seed coats, as discussed earlier for TMV. The low level of BPMV seed transmission, regardless of mechanism, may still provide a sufficient source of virus in the presence of high beetle populations to cause significant virus incidence. The potential impact of this low level of seed transmission is unclear because there is presently insufficient information available to assess the significance of this relative to various BLB populations.

Mottling of seed coats, similar to that observed with SMV, also occurs in soybean seed harvested from BPMV-infected plants. However, presence of seed coat mottling, as with SMV (9), is unreliable for predicting seedborne BPMV. One report, using a single soybean cultivar, suggests a positive linear relationship between percent mottling and amount of seedborne SMV (84). Recent work with other soybean cultivars demonstrates that either a positive or negative linear relationship between percent seed coat mottling and relative amount of seedborne BPMV can be demonstrated and that the relationship appears to be cultivar dependent in seed lots that were tested and found not to have SMV (J. H. Hill, *unpublished*). Further testing of single seeds from two soybean cultivars for presence of virus antigen has shown that in one cultivar, 31 of 35 nonmottled seeds contained BPMV while 47 of 65 mottled seeds tested positive. With the other cultivar, all 13 nonmottled seeds tested positive and 79 of 87 mottled seeds were positive (J. H. Hill, *unpublished*). It is conceivable that cultivars that exhibit a negative linear relationship between percent mottling and relative virus antigen content produce a relatively high number of seeds that contain virus antigen but are not mottled.

Overwintering BPMV in F_2 beetle population. Beetles transmit acquired BPMV as they deposit virus-containing regurgitant at feeding sites. Several lines of evidence suggest that beetle-transmissible viruses escape inhibition by RNase at beetle feeding wounds and that this differentiates them from viruses that are not transmitted by beetles. Beetle-transmitted viruses are transported in xylem more readily than non-beetle-transmitted viruses (25). In a preliminary study in Arkansas, Walters et al. (91) collected beetles during the winter months from trash in or near fields with high BPMV incidence the previous season. Although transmission efficiency was low (3%), Walters et al. (91) concluded that transmission was sufficient to establish sources of BPMV inoculum in volunteer soybeans and other available spring hosts. These authors also interpreted their data to indicate that BPMV overwinters in hibernating beetles, but they did not rule out that the beetles could have acquired BPMV by feeding on the underground parts of dormant plants. Unfortunately, the Arkansas observations made 30 years ago in an abstract have yet to be confirmed in a published journal article.

To study the significance of overwintering BLB as a source of primary BPMV inoculum in Kentucky (S. A. Ghabrial, *unpublished*), three approaches were used: (i) virus-containing BLB, as determined by ELISA of regurgitant, were collected from soybean fields with high BPMV incidence and placed in insect-proof cages for overwintering; (ii) BLB were collected from emergence traps that were placed during the winter in various locations in Kentucky with high virus incidence in the preceding season; and (iii) BLB were collected from alfalfa fields early in the spring (during April and May). Regurgitant was collected from all beetles and assayed for BPMV by ELISA. Virus-containing beetles were placed on healthy plants (one beetle per plant). The results indicated that ELISA readily detected BPMV antigen in regurgitant from a high percentage of the emerging beetles in both the cages and traps (40 positive beetles out of 80 beetles that survived overwintering, or 50%); however, none of the virus-containing beetles transmitted BPMV to healthy plants (0/40). Likewise, the viruliferous beetles collected from alfalfa fields (2 positive out of 107 beetles collected) failed to transmit BPMV to healthy plants. These overwintered beetles, however, regained ability to transmit virus following acquisition feeding on infected plants. However, a recent Iowa study indicates that a low percentage of BLB could transmit BPMV after emergence. In this study, leaf litter was collected from areas of high BPMV incidence in late fall and placed in outdoor cages. In spring, emerged beetles were collected from litter and placed singly on soybean plants grown from seed of virus-indexed greenhouse plants. Over a 2-year period in which 182 emerged beetles were tested, approximately 0.5% transmitted BPMV to soybean plants (R. K. Krell, *unpublished*). These results do not necessarily conflict with those obtained from Kentucky, as differences could result from different beetle biotypes, virus strains, or ambient temperatures during overwintering.

Management of BPMV with Vector Management

Controlling BLB populations is potentially an effective strategy to manage BPMV, particularly in the north central region where BLB is usually the only significant spring pest. Ross (70) showed that insecticide applied throughout the year reduced BLB populations to insignificant levels, preventing BPMV spread. Because infection after plant stage V6 has little effect on yield (71,96), control of the spring colonizing beetles may be sufficient to limit yield loss caused by BPMV.

Foliar insecticides. BLB is commonly controlled with foliar insecticides, which can be particularly effective on seedlings when insecticide coverage can be near complete. Because BLB transmit BPMV with minimal feeding, early treatment of the colonizing beetles is likely necessary to limit BPMV spread. Beetle colonization of soybean fields can begin at seedling emergence and continue through several seedling stages. Consequently, foliar insecticides should be applied early in the seedling stages and have residual action. Insecticide trials that target the colonizing beetles indicate they are susceptible to a variety of insecticides (19). Postemergence applied foliar insecticides allow feeding and oviposition of beetles prior to insecticide application.

Recently, the repellant properties of pyrethroids (15,16,31,74) have become the focus of BLB management with insecticides. Hammond (32) found that a single application of lambda-cyhalothrin provided long-term control of BLB if applied at the beginning of the F_2 generation beetle emergence. If used early in the seedling stages, lambda-cyhalothrin should likewise protect

soybeans from spring colonizing beetles. An advantage to foliar-applied insecticides is their therapeutic nature and the fact that they can be applied only when necessary.

Soil-applied systemic insecticides. Another option for BLB control is soil-applied systemic insecticides. Although not currently labeled for use for BLB on soybean, several systemic insecticides effectively reduce early season BLB numbers when applied in-furrow (carbofuran) (97) or as a band incorporated at planting (carbofuran, aldicarb, phorate, and disulfoton) (18). Advantages to using soil-applied systemic insecticides are efficacy upon plant emergence and possible larval toxicity. The disadvantage of soil-applied systemics is that they are preventative and do not allow the flexibility of not treating when colonization is minimal.

Systemic seed treatment insecticides. Seed treatments, like soil-applied systemics, offer the advantage of BLB control from plant emergence through seedling stages. These compounds are systemic and applied prior to sale. Initial studies with seed-applied imidacloprid and thiamethoxam indicate they effectively control BLB on seedling soybean (37). Seed treatments use very small amounts of active ingredient. However, as with soil-applied systemics, the decision to use this technology is made prior to BLB colonization (often in the previous year) and does not allow the flexibility of not treating if BLB colonization is low.

Although managing BPMV through vector (BLB) management appears promising, it does pose problems if insecticides, particularly soil-applied systemics and seed treatments, are used on a regional basis. Time and again, widespread use of a single management tactic to control insects has selected for resistance in the target pest population. Care must be taken to develop an integrated pest management approach to BPMV vector management. Because various insecticides, and indeed various cultural control options, are effective in reducing early season BLB, a rotation of tactics is recommended.

Planting date. Recent agronomic practice in the north central states has moved toward early planting of soybeans in many states. For example, Iowa statistics show that 50% of the acres were planted by 30 May in 1995 and by 5 May in 2000 (75,76). Although planting is delayed by weather conditions during some years, the trend toward early planting is associated with studies showing that, in the absence of BPMV, best yield usually results when soybeans are planted between late April and mid-May. Planting after mid-May often results in decreased yields in Iowa and many parts of the north central region (95). However, studies have shown that early planting favors increased BLB densities. Later planting reduces BLB densities and beetle colonization (Fig. 7A) (59,97). This is a function of colonization opportunity and beetle fitness (99). Bean leaf beetles typically emerge from overwintering habitat well before soybean emergence, so the first fields to emerge will attract many beetles simply because they are available. In addition, BLB fitness declines if soybean feeding is delayed, so delaying planting/emergence of soybeans increases precolonization mortality and reduces oviposition, resulting in lower BLB populations. Higher populations in early-planted soybean appear to relate to higher BPMV incidence based on preliminary studies in Nebraska (Fig. 7B).

Trap crops. Early-planted trap crops (56) are another possible BLB management option. Under this regime, portions of fields are planted early, concentrating BLB populations. Insecticides are then applied to the trap crop, effectively controlling beetles with minimal insecticide use. The disadvantage of this system is that machinery must be mobilized twice to plant one field.

Management of BPMV with Host Plant Genetics

Host plant genetics would be the most economical approach for the producer to manage BPMV. Upon infection, susceptible (virus readily infects and/or replicates and/or invades) or resistant (virus infection and/or replication and/or invasion is restricted) plant reactions show a range of tolerance or sensitivity in the plant (12). There are currently no commercial BPMV-resistant soybean cultivars. Resistance to BPMV has been identified in the genus *Glycine* (80) and may permit introduction of BPMV resistance with interspecific crosses. In 1985, four soybean germ plasm lines were released that were resistant to BPMV (72). Resistance to BPMV was determined by visual symptomatology. Symptomless lines were assumed to be resistant. As soybean lines can be infected without showing symptoms, it is possible that these are not resistant lines. However, based on what is known of other comoviruses, single gene resistance should exist. This is based on single gene resistance to the closely related comovirus, *Cowpea mosaic virus,* in *Vigna unguiculata* (L.) Walp. (63). Also, the virus genetic diversity identified to date (30) suggests the potential for corresponding host diversity based on what is known of other pathosystems. Screening of germ plasm will hopefully reveal these resistance genes. In the absence of true resistance, tolerance can be utilized. Tolerance levels vary in current germ plasm (33,100), and several differences in response have been identified among various germ plasm sources (34,55,64,70,79,96). It is critical that germ plasm evaluation consider virus strain diversity and monitor virus titer by ELISA since symptomology is not a reliable criterion for resistance (L. J. Giesler, *personal observation*).

Disease Management Through Pathogen Derived Resistance

In the absence of resistance in commercial soybean cultivars, researchers have resorted to transgenic resistance utilizing pathogen derived resistance (PDR). The concept of PDR, first proposed in 1985 (77), has been successfully utilized over the past 17 years to confer resistance against viruses in many crop plants. PDR involves the expression of viral genes in a host plant and the subsequent disruption of essential pathogenic processes of the challenge virus to confer resistance. PDR has been attained by expressing various forms of functional or dysfunctional viral coat protein, replicase, protease, and movement protein genes. PDR-mediated protection phenotypes range from delayed symptom development, reduced symptoms, and virion accumulation to apparent immunity. The variety of PDR phenotypes suggests multiple mechanisms underlying the attained resistance (4,21,22,46,52). The first example of PDR was in transgenic tobacco that accumulated coat protein (CP) of *Tobacco mosaic virus*; the resistance achieved was termed CP-mediated resistance (5). Protection against viruses with

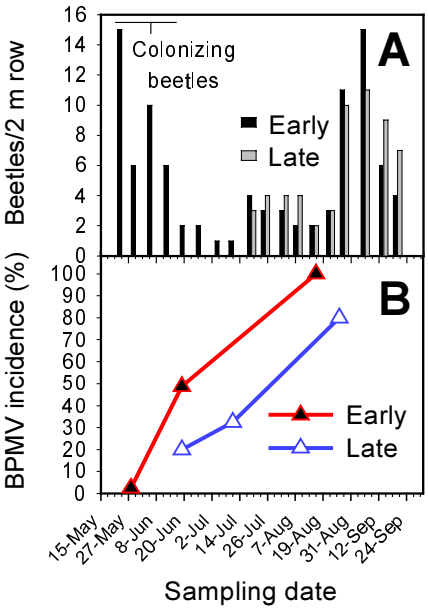

Fig. 7. A, Number of bean leaf beetles per 2 m of row for early (4 May) and late (26 May) planted soybean near West Point, NE, 1989. Adapted from Witkowski and Echtenkamp (97). B, *Bean pod mottle virus* (BPMV) incidence in early (19 April) and late (25 May) planted soybean near Fremont, NE, 2001. Foliage samples were collected at growth stages V2, V5, and R1 (L. J. Giesler, *unpublished data*). Percent incidence calculated by taking the average number of enzyme-linked immunosorbent assay (ELISA)-positive samples per strip (11 × 402 m). Ten pooled samples of six trifoliate leaves were collected per strip, with four replications of each planting date.

two coat proteins (such as comoviruses) could be achieved by expressing the capsid polyprotein (pCP) or by expressing both individual CP genes (14,57,58).

Di et al. (14) reported resistance to BPMV by expressing the pCP in transgenic soybean plants. However, the transgene utilized in that study was not stable, resulting in its loss in advanced generations of transgenic lines with a subsequent loss of resistance (S. A. Ghabrial, *unpublished data*). More recently, stable transformation of soybean with the BPMV pCP gene was achieved via particle bombardment of somatic embryo cultures. Resistance to BPMV (reduced symptoms and virion accumulation) appeared in the T_2 progeny of transgenic lines in plants inoculated mechanically and with viruliferous BLB (66). The features of pCP transgenic resistance (concentration of pCP in transgenic plants is correlated with resistance level) is reminiscent of coat protein-mediated resistance (5). Although the pCP transgenic lines provide valuable material that could be incorporated into commercial cultivars, further improvement of resistance level could still be attained via strategies known to confer complete resistance in other comovirus–host systems, e.g., expression of movement protein, replicase, or individual CP genes (58,81). The high level resistance reported in the latter studies appears to involve posttranscriptional gene silencing (PTGS). PTGS results in the degradation of RNA viruses, which have an RNA genome with nucleotide sequences similar to the transgene used for plant transformation (4).

Concluding Remarks

Fundamental and applied research on the epidemiology and host resistance to BPMV is urgently needed. Few disease management strategies have been developed or implemented for BPMV control, and no resistance to BPMV has been incorporated into commercial soybean cultivars. Comprehensive studies in several locations over at least three growing seasons are required to critically assess roles of seed transmission, overwintering beetles, and perennial hosts and/or alternate BPMV hosts as primary sources of BPMV inoculum. It is possible that all three potential sources of BPMV inoculum play more or less important roles in BPMV epidemiology dependent on location and environment. Seasonal variations that influence the survival and emergence of overwintering beetles as well as those that affect survival of alternate hosts and other putative virus reservoirs are expected to influence BPMV incidence.

The impact of strain diversity in BPMV is unclear. Each diversity group of BPMV isolates (genetically distinct strains, reassortants, recombinants) may have a different level of stability or virulence, as reflected by symptom severity in each variety of soybean. BPMV variants may also differ in seed transmission and ability to overwinter in beetles. Also, each diversity group may affect soybean production differently as environmental conditions vary. Until these parameters are examined further, it is hard to determine the effect BPMV will have on soybean production in the United States.

One of the first goals is to determine the potential yield reduction in current soybean cultivars due to BPMV infection. As per the evaluation of germ plasm and comparison among studies, it is critical that in all publications the plant stage of inoculation is noted and that the rating be done based on 100% plants infected compared with noninoculated or mock inoculated controls. This is best done in a split-plot arrangement to account for field variation among entries. It is also best to perform this test under field conditions with adequate moisture as opposed to greenhouse studies, because seed quality is normally poor under greenhouse conditions. In addition, field evaluations require the use of insecticides that have residual action as suggested in this article. It is also critical that the BPMV strain be identified based on current diversity characteristics, as mild and severe strains and reassortants are known to occur.

Through regional and interdisciplinary research projects, management strategies for BPMV will be developed. As this is a problem for pathologists, entomologists, and agronomists, a new regional project (NCR-200) has been established to foster linkages among all states and to integrate several areas of expertise. The North Central Soybean Research Program (NCSRP) has also initiated a substantial level of funding to support research and extension activities that will establish management guidelines for soybean viruses, including BPMV. These projects and individual state promotion board funds are collectively stimulating research in this area that will result in significant scientific contributions. These contributions will potentially lead to increasing the profitability and competitiveness of soybean producers in the United States by potentially increasing yields and seed quality.

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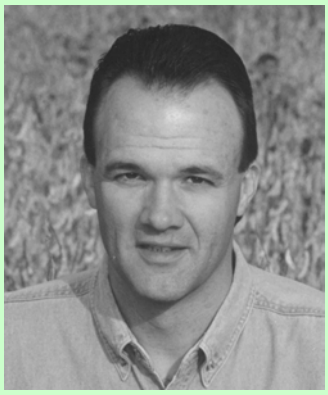

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