Peanut Mottle Virus Epidemics in Lupines

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ABSTRACT

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Blue lupine (*Lupinus angustifolius*) and white lupine (*L. albus*) became infected with peanut mottle virus (PMV) when planted next to peanuts. In each of 2 yr, more than 80% of lupines were infected with PMV. Incidence of PMV was very low in lupines planted more than 100 m from a virus source. Height reduction and yield losses appeared greater in lupines than occur in PMV-infected peanuts and soybeans. The rate of seed transmission of PMV in white lupine was less than 1%. Infected lupines can provide a vegetative overwintering source of PMV for the next season's peanut crop. Destroying peanuts before lupines are planted or planting lupines more than 100 m from peanuts may be an effective prevention of PMV epidemics in lupines.

Peanut mottle virus (PMV) has been recently reported to infect forage legumes including blue lupine (*Lupinus angustifolius* L.) and white lupine (*L. albus* L.) (3). In the winter of 1979–1980, a mixed planting of blue Tifblue-78 and white Tifwhite-78 lupines in Tift County, GA, had a high proportion of the plants apparently naturally infected with a virus. Diagnostic tests verified that these plants were infected with PMV. Other lupine plantings in this area did not appear to be infected to the same degree, indicating an opportunity to document a natural epidemic of PMV in lupines.

Previous reports have indicated that peanut seed is the primary source of PMV for agricultural crops in the Southeast (1,7,9). The virus spreads within this crop and then to adjacent susceptible crops (1,4). An overwintering vegetative source of PMV has not been documented. Although other crops, such as soybean (*Glycine max* (L.) Merr.), arrowleaf clover (*Trifolium vesiculosum* Savi), subterranean clover (*T. subterraneum* L.), blue lupine, and white lupine, are infected with PMV (3,4), reports of epidemics caused by PMV have not been documented except in peanuts.

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The purpose of this study was to: 1) determine the effect of natural PMV infection on blue and white lupine production, and 2) obtain information on the epidemiological factors that can lead to an epidemic. Important considerations were the possible transmissibility of PMV via seed and the feasibility that lupine could serve as a vegetative overwintering virus source.

MATERIALS AND METHODS

Virus identity. Leaf samples were collected from both blue and white lupines and assayed in the laboratory. Approximately 1 g of tissue was triturated with a mortar and pestle using 1 ml of 0.025 M potassium phosphate buffer (pH 7.2) containing 1% Celite. The buffered sap was used to mechanically inoculate *Phaseolus vulgaris* L. 'Topcrop,' a local lesion PMV indicator host, and *Pisum sativum* L. 'Alaska' or 'Little Marvel,' systemic hosts in which PMV attains a high titer. Tissue from infected peas was later used to inoculate *Glycine max* 'Bragg' and *Arachis hypogaea* L. 'Florunner' or 'Argentine' as additional diagnostic hosts. Identity of PMV was confirmed in pea sap by latex agglutination serological tests (6) in microcapillary tubes. Identity of PMV in blue and white lupines was also confirmed serologically.

Plot establishment. In the fall of 1979, plots of Tifblue-78 blue lupine and Tifwhite-78 white lupine were positioned as illustrated in Figure 1. Each plot consisted of alternating four rows of blue and four rows of white lupine, replicated eight times. The rows were 68 m long and the plots were 60 m wide. Plot A (next to peanuts) was planted on 4 October; plots B and C were planted on 18 October. The distance between plots B and C was 112 m.

On 14 February 1980, PMV-infected (visual symptoms) and healthy blue and white lupines were identified with plastic flagging. A total of 150 infected plants and 300 healthy plants were flagged for both blue and white lupines. Accuracy of visual diagnosis was confirmed by indexing about 5% of the plants on Topcrop bean.

On 9 April 1980, at the onset of flowering, flags were attached to plants that were infected on this date but appeared to be healthy in February. Plant heights were recorded at both times of flag attachment.

Peanuts were seeded in three spaced plots next to plot A during the summer of 1980 to again have a natural source of PMV for the fall-planted lupines. Blue and white lupines planted alternately in four rows were seeded between and adjacent to these peanuts in October. Height determinations and flagging of infected and healthy plants were as in the 1979–1980 lupine crop.

In the spring of 1981, while the lupines still had green foliage, peanuts were planted adjacent to and 85, 150, and 240 m from infected lupines.

Sampling methods and seed handling. Sampling to determine incidence of PMV was by visual inspection and inoculation of Topcrop bean from 20 consecutive plants, approximately 20 m apart, at four locations in each plot. In April, the number of winter-killed plants was recorded among the plants infected before February and the plants healthy in February.

In early June (pod maturity), seeds from individual plants of each group were collected, weighed, and sown in flats in the greenhouse to determine seed transmissibility of PMV. When seedlings were about 18 cm tall (fourth leaf stage), one leaf from each of five plants was

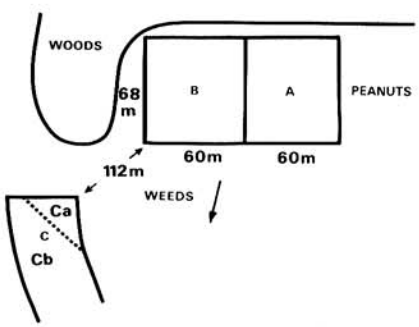

Fig. 1. Ecological setting for the lupine plots in 1979. Plot A was planted on 4 October and plots B and C on 18 October. Arrow indicates the prevailing wind direction.

pooled, triturated, and indexed by inoculation of Topcrop beans.

RESULTS

In February 1980, the incidence of PMV in the lupines was more than 40% in the plot 1–60 m from peanuts (Table 1), less than 30% in the area 60–120 m from peanuts, and not detectable in lupines 260 m from peanuts. By April, incidence of PMV in both blue and white lupines growing within 120 m of the original virus source in peanuts exceeded 79% infection. In February, incidence of the virus was less than 1% in lupines growing 230 m from the initial virus source and separated by 112 m from continuous lupine plants (Fig. 1, plot Ca) and 12–16% by April. Lupine plants growing 250 m from the initial virus source (Fig. 1, plot Cb) were not infected with PMV.

In December 1980, the incidence of PMV was 10% in both blue and white lupine in plot A, and reached 68% in the blue and 75% in the white lupine by February 1981.

Heights of PMV-infected blue lupine plants were reduced by more than 30% when infected early and by 20% when infected during midseason (Table 2). Height reduction of white lupine was less than 30% when infected early, and growth was not significantly different from the healthy checks or those infected later when measurements were made in April.

During the first week of March 1980, after spring plant growth was initiated, a severe freeze (−9 C) caused significant winterkilling among both blue and white lupines. Records taken during early April showed that 84% of the blue and 98% of the white lupines infected at midseason were winter-killed; 38% of the blue and 74% of the white lupines that were healthy at midseason were winter-killed.

Incidence of PMV in spring-planted peanuts that were adjacent to PMV-infected lupines was 70% higher than in peanut plants at 85 and 240 m from lupines, indicating that the virus spread from lupines to peanuts.

Based on measurements of randomly distributed plants, seed number and weight were reduced in PMV-infected blue lupines (Table 3). Both seed numbers and seed weight were reduced most by early infection.

Seed transmission of PMV in blue lupine was not detected with 331 seeds from plants infected early or with 960 seeds from plants infected during the month before flowering. In white lupine, one of 464 seedlings from seed of plants infected early and two of 335 from seed of plants infected during the month before flowering were infected with PMV.

DISCUSSION

For two consecutive years, lupines that were planted next to PMV-infected peanuts became infected with PMV, whereas lupines planted outside the peanut-growing areas were not infected with the virus. Initially, PMV incidence was higher in lupines nearest to peanuts, but later the virus spread through the contiguous lupine field. PMV incidence continued to increase in lupines several months after frost had eliminated peanuts as a source of virus, indicating that lupines can serve as a perpetuating source of virus for an epidemic. The incidence of PMV reached 80% in contiguous lupine plants located 100 m from the initial source (Fig. 1, plot B). The continual increase in virus incidence throughout the 2-yr period indicates that lupines are susceptible at all stages of growth and that PMV vectors are active in southern Georgia throughout the winter. Reported PMV vectors are the aphids *Aphis craccivora* Koch, *Rhopalosiphum padi* (L.), *Myzus persicae* (Sulzer), *Aphis gossypii* Glover, *Hyperomyzus lactucae* (L.), and *Rhopalosiphum maidis* (Fitch) (5), although none were observed colonizing lupines.

Lupines located more than 100 m from a virus source (Fig. 1, plot C) and separated by weed groundcover were infected but at a much lower level than plants that were contiguous to a virus source.

This is the first report of a high incidence of PMV in plants that were located more than 50 m from a virus source. In peanuts, the primary virus source is peanut seed (1,9), which provides a source of virus throughout the crop. When virus-free peanut seed is planted next to a virus source, a high percentage of the peanuts near the source is infected, but a low percentage is infected if the planting is 50 m from the source (1). Incidence of PMV in soybeans located next to PMV-infected peanuts may reach 25% but may be very low in soybeans 50 m or more from a virus source (1,4). The high incidence of PMV in lupines located 100 m from a virus source may be due to

Table 1. Incidence of peanut mottle virus in blue and white lupines at different times during the growing season and at different distances from an initial virus source in peanuts

Distance from virus source	Percent infection (1980)	
	February	April
1–60 m		
Blue	48	86
White	43	81
60–120 m		
Blue	28	84
White	19	79
230–260 m		
Blue	< 1	16
White	0	12
> 260 m		
Blue	0	0
White	0	0

Table 2. Effect of peanut mottle virus on the height of lupines in 1980

Cultivar	Height (cm)[y]	
	14 February	7 April
Tifblue-78		
Infected before 14 February	24 a	43 c
Infected between 14 February and 7 April	...	51 b
Healthy	35 b	64 a
Tifwhite-78		
Infected before 14 February	14 b	38 a
Infected between 14 February and 7 April	...	44 a
Healthy	17 a	40 a[z]

[y] Based on an average of 24–150 plants, depending on availability of plants in the entry group. Numbers followed by the same letter for a cultivar are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

[z] Data based on only three plants because of winterkill, rabbit damage, and high percentage of infection.

Table 3. Yields of blue and white lupines infected with peanut mottle virus in 1981[y]

Cultivar	Average number of seed per plant	Average yield (g) per plant	Average weight (mg) per seed
Tifblue-78			
Infected before 5 February	40.7 b	3.4 c	95 b
Infected between 5 February and 20 March	67.7 b	7.4 bc	95 b
Infection after 20 March	114.3 a	13.3 a	126 a
Tifwhite-78			
Infection before 5 February	7.3 a	1.3 b	157 a
Infection between 5 February and 20 March	16.7 a	3.4 b	199 a
Infection after 20 March	16.9 a	3.6 b	189 a
Healthy[z]	32.8 a	10.5 a	323 a

[y] Numbers followed by the same letter for a cultivar are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

[z] Plants of the same cultivar but growing more than 1 km from infected plants.

their lack of resistance as they age and to their long growing season (October–June).

Our results show that lupines can serve as a source of PMV for peanuts and as a vegetative overwintering host of the virus.

Although the rate of seed transmission of PMV in white lupine was low (0.37%), it could be significant. Seed transmission in peanuts is often less than 1% (9), yet field spread can reach epidemic proportions (1,9). Because lupines are a long-season crop that does not become resistant with age, and aphid vectors are present throughout the season, an initial seed infection of less than 1% could result in significant spread of the virus.

PMV causes mild symptoms in peanuts, (7) with little stunting and mild symptoms in cowpeas (Demski et al, *unpublished*), and it affects soybeans only slightly (2). In lupines, however, PMV causes more severe effects. Studies of losses in lupines to PMV are hampered in the field by the high levels of virus spread and in the greenhouse because a cold period is needed for normal growth and proper seed maturation. Statistical analysis of lupine yield reduction is further hampered by a high percentage of winterkill of infected plants and a high incidence of disease, leaving few healthy plants for comparison. Therefore, the yield reduction caused by PMV in lupines reported in this paper is considered preliminary. Our observations, however, indicate that loss is much greater in early-infected lupines than in peanuts and soybeans, where a 20% loss in both is common (2,8).

When lupines are planted in the peanut belt in Georgia, one effective control measure for PMV may be to delay planting until after frost kills vegetative peanuts that can provide a source of the virus for lupines or to plant lupine more than 100 m from peanuts. Numerous white and blue lupine plantings were within 5 km of the documented PMV lupine epidemics. None of these plantings developed a high incidence of PMV. Epidemics occurred only when early-planted lupines (Fig. 1, plot A) were near PMV-infected vegetative peanuts.

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