

Source and Spread of Peanut Mottle Virus in Soybean and Peanut

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ABSTRACT

Virus incidence and distribution data show that the source of peanut mottle virus for soybean is infected peanut. The source of virus in peanut is infected seed, confirming a previous report. The virus is transmitted from peanut to soybean, from soybean to soybean, and from soybean to peanut. The virus can move at least 48 m in a single step to previously uninfected plants, but apparently not 6.5 km. As the distance between the inoculum source and adjacent

plantings increases, the time until initial infection increases, resulting in a lower percentage of disease at crop maturity. The rapid increase of this disease in a planting was the result of inoculum dissemination from within, as opposed to between, plantings. It is this internal spread that leads to epiphytotic conditions.

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Additional key words: aphid trapping, *Glycine max*, virus movement.

Peanut mottle virus (PMV) naturally infects both soybean, *Glycine max* (L.) Merrill (1, 2, 6), and peanut, *Arachis hypogaea* L. (1, 2, 5), in several areas of the world. The virus causes significant yield reduction in soybean (4) and peanut (5). The economic loss in soybean from PMV infection has not been determined; however, the loss in peanut in Georgia in 1973 was estimated to be over \$10 million (8).

In 1974, Paguio and Kuhn (9) reported the source of PMV inoculum for peanut was peanut seed by showing that PMV was seed-transmitted in peanut, that it could not be isolated from weed hosts, and by using van der

Plank's test for adjacent (paired) plants to study internal spread.

Bock (2) reported no PMV seed transmission in soybean, and Demski and Harris (3) showed by assaying a total of 5,929 seed of three cultivars (Bragg, Hampton 266A, and Jackson) that the virus did not pass through the seed. In addition, no evidence is indicated for soil transmission.

The purpose of this study was to investigate the source of PMV for both soybean and peanut by utilizing susceptible soybean and virus-free peanut seed, and to show the disease gradient and rate of disease increase as a

function of proximity of the virus source. In this paper, progressive spread means the spread of virus within or through a plot during the season and single-step spread means spread between plots.

MATERIALS AND METHODS.—*Inoculum source.*—Field surveys for PMV naturally infecting soybean were conducted in 1971, 1972, and 1973 in Georgia. Identification of PMV was based on indexing to the diagnostic local-lesion bean host (*Phaseolus vulgaris* L. 'Topcrop'), visual observations of symptoms in soybean and peanut, and occasional checks on the physical and serological properties in crude sap.

Inoculum was the mild strain (M-2) of PMV which is the most common in soybean and peanut (6, 7). The isolate was obtained from C. W. Kuhn (7). This isolate was maintained in garden pea, *Pisum sativum* L. 'Little Marvel'. Mechanical inoculations were made with infected pea leaves ground in 0.05 M phosphate buffer (pH 7.5) containing 0.01 M NaHSO₃ and 1% Celite.

To determine the effect of volunteer peanut plants on the establishment and spread of PMV in soybean plantings, two 0.10-hectare (ha) plots of peanut with greater than 75% PMV infection in 1972 were not harvested. The next year these plots, and an additional 0.20 ha of adjacent land, were disked and planted to soybean. In addition, seeds harvested from the same soybean lot were planted 50 m distant from the original plots.

To obtain PMV-free peanut seed, commercial lots of cultivars Argentine and Florunner were grown in the greenhouse. The seedlings were individually indexed to

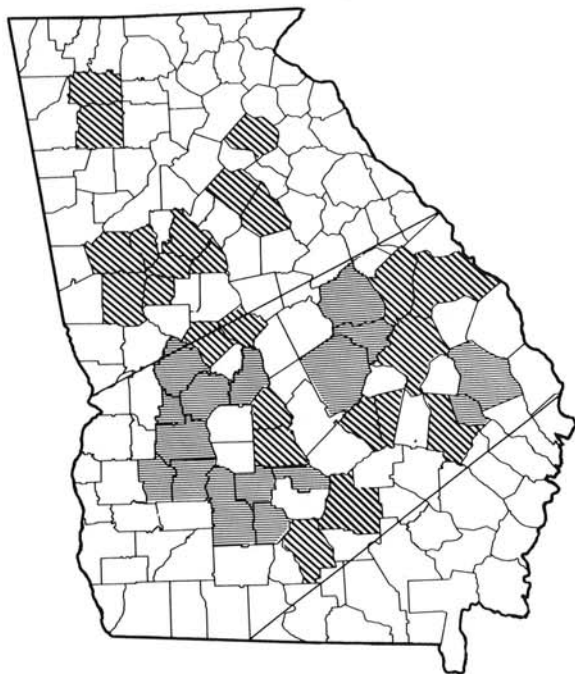


Fig. 1. Distribution of peanut mottle virus naturally infecting soybean in Georgia. Virus was not recovered in diagonally lined counties, and was recovered from the shaded counties. Over 95% of the Georgia peanuts are grown in the area between the two lines drawn across the map.

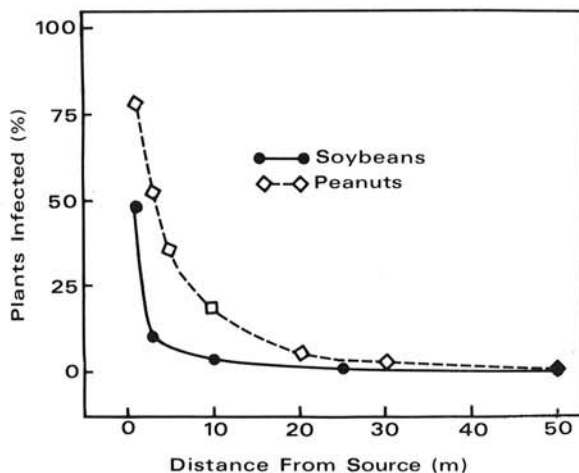


Fig. 2. Spread of peanut mottle virus in soybean and peanut from a single-row source located between the two plots. Data represent the percentage of plants infected 70 days after a virus source was provided.

Topcrop bean. Infected peanut seedlings were rogued and healthy seedlings were grown to maturity. Before harvest, all plants were again indexed to Topcrop. Seed from these virus-free parent plants were planted on land with no history of peanut production and located over 6.5 km from the nearest known peanut field. These field-grown peanuts were indexed to Topcrop at midseason, and again just before harvest.

Spread from crop to crop.—Virus movement within or between soybean and peanut was determined by various field plot arrangements from 1971 through 1974. Plots were located a minimum of 100 m apart. To establish infection in experimental plots, specific plants were mechanically inoculated when the first trifoliolate leaves were fully expanded. Counts of PMV-diseased plants were made utilizing field symptoms and indexing to Topcrop bean.

One experiment had four rows of Bragg soybean placed beside a triangular shaped plot (53 × 53 × 30 m) of corn with four rows of peanut (inoculated with PMV) along a second side. Counts of PMV-infected soybean were made weekly until 3 weeks after flowering.

Two plots, one with Argentine peanut and the other with Bragg soybean, were positioned next to each other. Each plot had 20 rows (1 m between rows) that were 30 m long. Peanut plants in the row closest to soybean were inoculated with PMV. PMV-diseased plants were counted in each row of both plots. In another test, the same arrangement was used except that the soybean plants closest to peanut were inoculated.

Bragg soybean (22 rows, 50 m long) was grown in isolation from peanut plots. All plants in the 11th row were inoculated with PMV. Percentage infection was determined based on distance from the inoculated row.

PMV-free peanut seed were planted in four isolation plots (10 rows, 10 m long) within a large soybean field. The peanut plots were 50 m from each other. Soybeans around two peanut plots were inoculated with PMV, whereas those around the other plots were not inoculated. Four weeks after symptoms developed, periodic

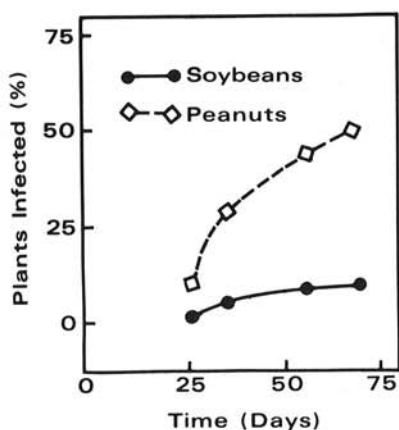


Fig. 3. Spread of peanut mottle virus in soybean and peanut from a single-row source located between the two plots. Data represent the percentage of plants infected 3 m from a virus source.

examinations were made for virus infection in the peanut plots.

Progressive and single-step spread.—The progressive development of virus infected plants from a known source of inoculum was determined in adjacent soybean and peanut plots. Each plot had 50 rows 18 m wide. Virus-free seed were used in these studies. The first row in each plot was inoculated with PMV, and the percentage plants infected in relation to time and distance from the single row source was determined.

The distance that PMV spreads in individual steps was measured by planting a small peanut plot (6 rows, 10 m long) in the center of a fallow field. One peanut plot (4 rows, 10 m long) was located in each of the four quadrants. These were 6, 12, 24, and 48 m, respectively, from the central plot. The two outside rows of the central plot were inoculated.

Aphid activity.—Winged aphid activity was monitored in the plots with yellow-pan water traps positioned 20 cm above the ground. Aphids were collected and traps were cleaned twice a week.

RESULTS.—*Inoculum source.*—Surveys showed that PMV was present in soybeans in 16 of 40 counties examined (Fig. 1). PMV was recovered from soybeans only in the peanut belt, the area of Georgia in which over 95% of the peanuts are produced. During the surveys, PMV was isolated from plants in 67 soybean fields; sixty-three were located near peanut fields. Of the remaining four fields, two had volunteer peanut plants from the previous year, and in two fields no volunteer peanut plants were observed in the vicinity. In the peanut belt, 43 soybean fields with no peanut in proximity were apparently free of PMV and six soybean fields next to peanut were apparently free of PMV. The latter fields could be free of PMV because some soybean cultivars are known to be resistant to PMV (4). Outside the peanut belt, 88 soybean fields were free of PMV, and none of these was near peanut. A survey (8) of peanut fields showed that PMV was present in each of 117 fields, thus all peanut fields may be a potential source of PMV inoculum for soybean.

Initially, about 1% of the soybeans planted in plots where volunteer peanuts occurred developed virus

symptoms, whereas adjoining plantings without volunteer peanuts remained virus free. Subsequently, the virus did spread to the adjoining soybeans that had no volunteer peanut plants. The final percentage of PMV-infected soybeans was 2%, and infection was restricted to plants immediately adjacent to peanut volunteers. Plants in the soybean plot 50 m distant did not become infected.

It was established in 1973 and 1974 that virus-free peanut could be grown in an isolated area in Spalding County (outside the peanut belt). When virus-free seed were obtained in the greenhouse and were used for planting a 0.2-ha plot in 1973, no plants were observed with PMV symptoms, and no virus was detected in 300 plants indexed to Topcrop bean. Seed from the 1973 plot were planted in the same area in 1974 with the same results. Furthermore, the 1973 seed were used in other experimental plots, and there was no evidence of seed transmission. When commercial peanut seed were planted in Spalding County in 1973 and 1974, there was an abundance of PMV-infected plants.

Spread from crop to crop.—Using mechanically inoculated plants as the only source of inoculum, it was clearly established that either peanut or soybean can serve as a source of inoculum for itself or the other crop.

A triangular corn buffer between infected peanut and healthy soybean was effective in demonstrating that PMV spreads from peanut to soybean. Initially, only the soybeans located 3 m or less from peanuts became infected, but in time the virus was isolated further from the source. At maturity, the greatest percentage infection in soybean was nearest peanut (8.2%), whereas plants 25 m distant had less than 1% infection. In addition, three other tests (J. W. Demski, *unpublished*) showed that soybeans closest to infected peanuts had the highest percentage of infection, indicating the virus spreads from peanut to soybean.

Peanut plants from virus-free seed grown beside soybean, or in isolation plots within soybean fields, became infected if the adjacent soybean had PMV. The peanuts nearest to infected soybeans became infected first. Peanuts in isolation plots first exhibited PMV symptoms four weeks after soybeans near them were inoculated and at maturity the percentage of infected peanut plants had increased to over 50%. In contrast, the isolated peanut plots with healthy soybean around them did not become infected until near maturity (probably by progression through soybean) and the final percentage infection was 2%. Thus, the virus can move from soybean to peanut.

When the center row in a soybean plot was infected with PMV, the virus infected 37% of the plants in the rows

TABLE 1. Movement of peanut mottle virus to peanut over a fallow field after a virus source was provided^a

Distance from source (m)	Time until first infection (days)	Final infection (%)
1	14	>80
6	21	77
12	21	38
24	36	14
48	44	7

^aA virus source was centrally located so that one plot (either 6, 12, 24, or 48 m distant) was in one of the four quadrants.

closest to the inoculated row, and less than 4% 10 m from the source, indicating that the virus spreads naturally from soybean to soybean.

Progressive and single-step spread.—From a single-row source (over 80% of the plants infected) virus progressed across a 50 m peanut field during the course of the growing season. One meter from the source there was 80% infection and 1% infection 50 m from the source (Fig. 2). Progressive spread of PMV was more rapid and produced a higher percentage infection in peanut than in soybean (Fig. 3) in the same period of time.

Spread of PMV in individual steps showed the virus was carried 6, 12, 24, or 48 m at one time (Table 1). Generally, the virus spreads quickly over the shorter distances. There was a close relationship between distance from the original source and percentage infection. Those plants located a greater distance from the source had a lower percentage of virus infection at maturity.

Aphid activity.—Aphids have been reported to be vectors of PMV (2, 4); however, a negative relationship was observed between winged aphid activity and virus spread in experimental plots over a 3-year period. At the time when natural spread occurred in soybean and peanut, the winged aphid population was low and at some periods, no winged aphids could be trapped. Infection of soybean and peanut with PMV occurred at the same rate both during times when aphids were trapped and when no aphids could be trapped.

DISCUSSION.—The reported percentages of PMV transmission through peanut seed have been variable. Kuhn (5) reported PMV seed transmission of 2%, Sun and Hebert (10) 0.001%, Bock (2) 20%, and Paguio and Kuhn (9) 0.3%. Although variable, this evidence clearly demonstrates a potential source of virus that may be distributed within field-planted peanuts. The same potential does not exist in soybean because PMV is not transmitted through the seed (2, 3) and no evidence is known for soil transmission. In addition, PMV could not be recovered from weeds, trees, shrubs, or vines in or near peanut fields (9). Therefore, the use of susceptible virus-free soybean is an excellent tool in demonstrating the virus source.

Field plot studies and virus disease surveys demonstrate a relationship between peanut and PMV-infected soybean. Results indicate that peanut is the source of virus for soybean in Georgia. This is supported by the fact that PMV in soybean has been found only in the peanut-growing area of the state.

Since peanut was grown to maturity free of PMV (using virus-free seed) in isolation from other peanut, it supports a previous report by Paguio and Kuhn (9) that the source of PMV is peanut seed. They also reported that PMV could not be recovered from weed hosts, and this study supports that conclusion. If weed hosts were a source of PMV, susceptible soybeans outside the peanut belt should become infected. Likewise the isolated virus-free peanut should have become infected. If weeds restricted to the peanut area are reservoir hosts, then soybeans grown in the peanut belt away from peanut should become infected; however, this was not the case in these studies. Since the virus spreads in peanut and soybean outside of the peanut belt (test plots in Spalding and Gordon Counties), it demonstrates that a vector of PMV is present state-wide. Thus, if a PMV reservoir is

near, soybean should become infected.

These studies suggest that epiphytotics resulting in significant yield losses are due to virus spread over short distances in soybean and peanut. Thus, spread within a field is important, and the time at which the virus appears in the plantings and the amount present early in the season is critical. Longer distance spread (48 m or more) does occur, but at shorter distances from the source, the time required for infection decreased and the percentage plants infected at maturity increased. It appears that the virus can spread more than 48 m in a single step, but not as far as 6.5 km.

Since PMV spreads faster and farther in peanut than soybean, with the same amount of inoculum present, it appears there is a vector preference (or greater efficiency) for peanut. However, even though the identity of all aphid species was not determined, both the number of aphids and species trapped were about the same in the two crops. Although a vector could affect the incidence of disease, it would not account for zero infection of soybean when it is planted in the peanut belt, but not close to peanut. Furthermore, a previous report showed soybean becomes resistant to PMV with age (4), which may account for a lower disease level at maturity, but it would not account for less infection in soybean when both crops are young and fully susceptible (Fig. 2).

The low aphid population during periods of virus spread suggests either that the aphids transmitting the virus are very efficient, or that perhaps another vector is involved. PMV belongs to the PVY group which is aphid transmitted. This does not rule out other PMV vectors, but none has been found to date. A previous report (4) showed that aphids transmit PMV more efficiently in peanut than in soybean. This is in accordance with the results of this paper. Nevertheless, this does not account for virus spread during periods when no aphids were trapped. Further studies about vector transmission of PMV in the field are needed.

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