A Virus Causing Peanut Mild Mottle in Hubei Province, China

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ABSTRACT

Xu, Z., Yu, Z., Liu, J., and Barnett, O. W. 1983. A virus causing peanut mild mottle in Hubei Province, China. Plant Disease 67: 1029-1032.

A virus that causes mild mottle on peanut is prevalent in the peanut-growing areas in the Hubei Province of China, where infected plants ranged from 1 to 98% in 20 fields surveyed in Hongan County. Soybean plants in fields near peanuts were also infected. The virus reduced peanut and soybean yields by as much as 23 and 53%, respectively, in greenhouse trials. Of 38 species of plants tested, 10 were infected. The virus was readily transmissible by sap-inoculation. Two species of aphids, *Aphis craccivora* and *Myzus persicae*, transmitted the virus in a nonpersistent manner. Transmission was 1.3 and 4% in seed collected from two peanut cultivars in severely infected fields and 4.8% in seed from plants infected by sap-inoculation. Longevity of the virus in vitro was 4–5 days, thermal inactivation point 55–60 C, and dilution end point 10^{-4}–10^{-5}. Virus particles were flexuous rods 773 nm long. Scroll-type pinwheel inclusions were found in cytoplasm of leaf tissue of both *Nicotiana benthamiana* and *Trifolium incarnatum*. The virus was serologically distantly related to peanut mottle virus and bean yellow mosaic virus. Molecular weights of the virus coat protein subunit and RNA were 33,500 and 3×10^6, respectively, by polyacrylamide gel electrophoresis. On the basis of host range, transmission characteristics, in vitro properties, particle and inclusion body morphology, serology, and physical and chemical properties, the potyvirus that causes peanut mild mottle was different from other peanut viruses. Of 633 peanut germ plasm lines tested, more than 80% of plants of most lines were infected and no peanut line was resistant.

A virus disease distinguished by a mild mottle on leaves of infected peanut plants (*Arachis hypogaea* L.) was found on the Oil Crops Research Institute's farm in the early 1960s but attracted little attention because crop loss was apparently slight. In a 1981 survey, however, a high incidence of the disease was recorded on the institute's farm and in some peanut fields of Hongan County, which is the main peanut-growing area in Hubei Province. On the basis of host range, transmission characteristics, in vitro properties, particle and inclusion body morphology, serology, and physical and chemical properties, the new potyvirus that causes peanut mild mottle disease (VPMM) is different from other peanut viruses.

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The first author was a visiting plant pathologist in the Department of Plant Pathology and Physiology, Clemson University, Clemson, SC 29631, when part of this research was done.

Accepted for publication 26 May 1983.

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MATERIALS AND METHODS

A survey of incidence of peanut mild mottle disease was carried out on the Institute's farm and in Hongan County of Hubei Province in 1981. Three hundred to 500 randomly chosen plants were examined in each field. The number of infected plants was based on visual inspection for symptoms.

Yield reduction of Red Flower 1 peanut and 1138-2 soybean (*Glycine max* (L.) Merr.) caused by VPMM was assessed in the greenhouse. One group of plants was sap-inoculated by grinding infected plant tissue in 0.07 M potassium phosphate buffer, pH 7.0, containing 0.02 M 2-mercaptoethanol and wiping on Carborundum-dusted leaves. The leaves were rinsed with tap water after inoculation. Another group of plants was maintained free of virus as a control.

A virus isolate from a single peanut plant with typical symptoms was chosen for characterization. The host range of the virus was determined by inoculating 10 plants of each species in the seedling stage. Inoculated plants were observed for 1 mo for symptoms. Plants with no symptoms were assayed by inoculation to soybean 1138-2 or *Chenopodium amaranticolor* Coste & Reyn.

Peanut seeds were collected from two cultivars in a field with high incidence of virus infection and from plants infected by sap-inoculation to check for seed transmission. The seeds were sown, grown in a greenhouse, and the number of seedlings with mottle recorded.

Tests for virus transmission by aphids were done by conventional methods with *Aphis craccivora* Koch and *Myzus persicae* Sulzer. Aphids were starved for 2–3 hr, then placed on virus-infected soybean leaves for 2 min. Aphids were transferred to healthy plants for 24 hr, then killed with an insecticide. More than 10 aphids were placed on each healthy plant. Aphids placed on healthy soybean leaves were also transferred to healthy plants as a check.

Tests of in vitro properties of the virus included longevity in vitro, thermal inactivation point, and dilution end point. Infected soybean leaves were used as a virus source. Infected tissue was ground in 0.07 M potassium phosphate buffer, pH 7.0, containing 0.02 M 2-mercaptoethanol. Soybean 1138-2 was used as the indicator plant. Sap was stored at 15 C for longevity in vitro tests.

For purification, the virus was propagated in *Nicotiana benthamiana* Domin. Purification was by the method devised by Sun and Hebert (15) but we used 0.5 M sodium citrate with 1% 2-mercaptoethanol and 0.5 M urea, pH 7.8, as the grinding buffer. Further purification was obtained by a cesium sulfate density gradient run for 17–18 hr at 73,500 *g*. Purified virus in 0.03 M sodium phosphate buffer, pH 7.0, was mounted on carbon-coated collodion membranes, stained with 2% ammonium molybdate, pH 7.5, and viewed in an electron microscope. Particle size was determined by measuring 100 virus particles. A grating replica of 2,160 lines per millimeter was used to calibrate magnification.

To evaluate inclusion bodies, *N. benthamiana* and *Trifolium incarnatum* L. with prominent symptoms were fixed in 3.5% glutaraldehyde in 0.1 M cacodylate buffer, pH 7.4, postfixed in a 1:1 solution of 2% osmium tetroxide and cacodylate buffer, dehydrated in a graded ethanol series, and embedded in Spurr's low-viscosity medium. Thin sections were cut, stained with 1% uranyl acetate followed by lead citrate, and observed.

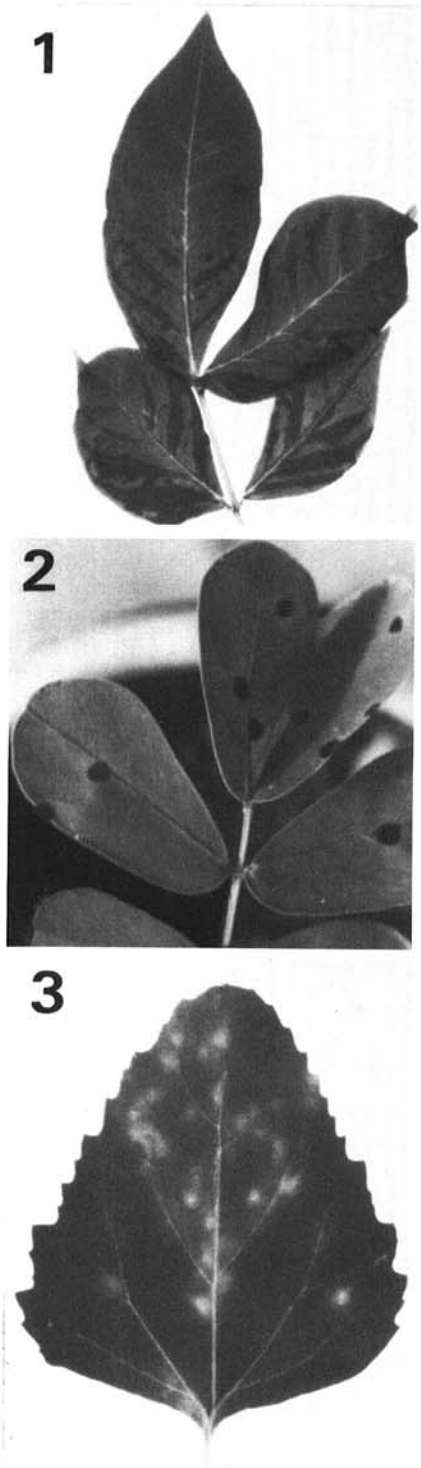
Antiserum to PMV, pea seedborne mosaic virus, and turnip mosaic virus were provided by S. A. Tolin, R. O.

Hampton and R. Shephard, respectively. Antisera to potato virus Y (PVAS 50) and tobacco etch virus (PVAS 69) were from the American Type Culture Collection, Rockville, MD. Other antisera were from laboratory stocks at Clemson, SC. Gel double-diffusion and enzyme-linked immunosorbent assay (ELISA) serological tests were used; 0.8% agarose with 0.1% sodium azide and 0.5% SDS in 0.03 M sodium phosphate, pH 7, was used as the medium for gel double-diffusion tests and previously established procedures were used for ELISA (9). Sap extracted from infected *N. benthamiana* leaves with prominent VPMM symptoms was used in all serological tests. Peanut mottle virus was kindly provided by C. W. Kuhn.

Molecular weight of virus coat protein was analyzed by electrophoresis in 12.5% polyacrylamide gels by the method of Laemmli (8). The following proteins were used as standards: α-lactalbumin (mol wt 14,400), soybean trypsin inhibitor (mol wt 20,100), carbonic anhydrase (mol wt 30,000), ovalbumin (mol wt 43,000), bovine serum albumin (mol wt 67,000), and phosphorylase b (mol wt 94,000).

For extraction of RNA, freeze-dried virus was resuspended in a small volume of 0.5 M Tris-HCl, pH 9. An equal volume of a solution containing 0.2 M $(NH_4)_2CO_3$, pH 9, 0.002 M Na_2EDTA, and 2% SDS was added to the virus, which was then layered on step gradients made of 8.5 ml of 30%, 8.5 ml of 22.5%, 8.0 ml of 15%, and 7.5 ml of 7.5% sucrose in 0.5 M Tris-HCl, pH 9. The gradients were centrifuged at 66,000 *g* for 16 hr at 14 C, then fractionated with an ISCO model D density gradient fractionator. The RNA was recovered by adding several volumes of ice-cold ethanol, then 3 M sodium acetate, pH 5.6, to a final concentration of 0.1 M, incubating overnight at −18 C, and centrifugating at 12,100 *g* for 30 min. Molecular weight was determined by electrophoresis in 1.8% acrylamide gels containing 0.5% agarose in a 0.04 M Tris, 0.02 M sodium acetate, 0.001 M Na_2EDTA, pH 7.2 buffer, run at 5 mA for 3 hr. Standards used were TMV-RNA (mol wt 2.05×10^6), *E. coli* 23S RNA (1.07×10^6) and *E. coli* 16S RNA (0.55×10^6). Ultraviolet absorption (260 nm) profiles were obtained with a GCA-McPherson spectrophotometer equipped with a gel-scanning attachment.

Tests of peanut germ plasm lines for resistance to VPMM were carried out in the field on the Institute's farm. Each line was grown in two or three rows with about 30 plants per line. A count of infected plants in each line was conducted in late June, when the virus was prevalent in the field.



Figs. 1–3. Symptoms of virus causing peanut mild mottle. **(1)** Mosaic on *Cassia occidentalis*, **(2)** black necrotic lesions on *C. obtusifolia*, and **(3)** chlorotic local lesions on *Chenopodium amaranticolor*.

RESULTS

Field survey and yield losses. Peanut plants with mild mottle symptoms were found in the plastic-mulched plots on the Institute's farm as early as 12 May. The incidence of infected plants was 2.3% in the field on 28 May but as high as 50.8, 86.4, and 100% on 9 June, 15 June, and 10 July, respectively. The top leaflets of infected plants frequently had distinct chlorotic spots or ring spots. After several days, the chlorotic spots and ring spots disappeared and the leaves showed typical mottling with dark green islands on a light green background. Diseased plants were not noticeably stunted. In July and August, when high temperatures prevailed, most infected plants were symptomless but some infected plants still were mottled.

Symptoms on infected peanut plants in fields of Hongan County were identical to those on the Institute's farm. Percentages of infected plants in 20 fields ranged from 1 to 98 (average 46%). In general, early-sown peanut fields had higher percentages of infected plants than late-sown ones. The incidence of infected plants was as high as 98% in a field of peanut mixed with cotton where a high density of aphids occurred.

Soybean plants near peanut fields were also infected by the virus. Infected soybean plants showed systemic mosaic symptoms. A sample from soybean on the Institute's farm and four samples from soybean and peanut collected in Hongan County were assayed by sap-inoculation to peanut, soybean, *C. amaranticolor*, and Topcrop bean. Symptoms on these indicator hosts were the same as those caused by the peanut isolate studied in detail.

Healthy peanut plants averaged 2.08 g dry pods per plant (36 plants), whereas infected plants averaged 1.59 g (28 plants) for an average yield loss of 23% caused by the virus. Healthy soybean plants averaged 3.83 g seed per plant (30 plants), whereas infected plants averaged 1.8 g seed per plant (30 plants), for an average yield loss of 53%.

Host range of VPMM. The virus had a narrow host range comparable to many potyviruses. Of 38 species of plants inoculated, the following 10 became infected:

Peanut Red Flower 1: top leaflets showed chlorotic spots and ring spots 5–6 days after inoculation. As chlorotic spots and ring spots disappeared, the leaves developed a light and dark green mottle. At high temperatures, symptoms on some infected plants were not apparent but were still visible on others. Infected plants were not stunted.

Soybean 1138-2, Aijiaozao: newly emerging leaflets showed veinclearing 5–6 days after inoculation. As veinclearing disappeared, mosaic developed on the leaves.

Cassia occidentalis L.: veins of newly emerging leaflets appeared faint and wider 10 days after inoculation and chlorotic spots developed along the veins. Systemic mosaic symptoms (Fig. 1) developed later.

C. obtusifolia L.: black necrotic lesions appeared on the inoculated leaves after 4 days (Fig. 2). As inoculated leaves turned yellow and dropped off the plant, necrotic rings developed on the upper leaves.

Trifolium incarnatum L.: top leaflets developed mosaic 10 days after inoculation.

N. benthamiana and *N. clevelandii* Gray: upper leaflets developed mosaic 7 days after inoculation.

C. amaranticolor: 1 wk after inoculation, chlorotic local lesions developed that changed to white necrotic lesions with a reddish brown halo 3–4 mm in diameter (Fig. 3). No systemic symptoms developed.

C. album L. and *C. quinoa* Willd: chlorotic local lesions developed 5 days after inoculation. No systemic symptoms developed.

The following species were not infected by the virus (no virus was recovered by inoculating soybean 1138-2 or *C. amaranticolor*): *Vigna unguiculata* subsp. *sesquipedalis* (L.) Verdc. 'Hongzuiyan' and 'Baitiaoxian'; *V. unguiculata* subsp. *unguiculata*; *Phaseolus vulgaris* L. 'Royal Red,' 'Topcrop,' and 'Tamada'; *P. angularis* (Willd) W. F. Wright; *P. mungo* L.; *Vicia faba* L.; *V. sativa* L.; *Dolichos lablab* L.; *Pisum sativum* L.; *Sesbania cannabina* Poir.; *Melilotus albus* Desr.; *N. rustica* L.; *N. glutinosa* L.; *N. tabacum* L. 'Xanthi-nc'; *Lycopersicon esculentum* Mill.; *Hibiscus sabdariffa* L.; *Physalis floridana* Rydb.; *Datura stramonium* L.; *Cucumis sativus* L.; *Zinnia elegans* Jacq.; *Petunia hybrida* Vilm.; *Spinacia oleracea* L.; *Beta vulgaris* L.; *Phaseolus lunatus* L.; *Trifolium hybridum* L.; *T. repens* L.; *T. pratense* L.; and *Glycine max* 'Bragg,' 'Rampage,' 'Bansei,' and 'Davis.'

Transmission of VPMM. The virus was readily transmitted by mechanical inoculation. The virus was transmitted to 3 of 6 peanut plants by *Aphis craccivora* and to 10 of 11 soybean plants by *Myzus persicae*. All check plants remained symptomless.

Of 151 plants of Fuhua Sheng peanut grown from seeds collected in a field with high incidence of virus infection, two (1.3%) were infected; of 151 plants of Red Flower 1 peanut, six (4%) were infected. Of 125 plants of Red Flower 1 peanut from seeds of sap-inoculated plants, six (4.8%) were infected.

In vitro properties of VPMM. Infectivity survived storage at 15 C for 4 but not 5 days, heating for 10 min to between 55 and 60 C, and dilution to between 10^{-4} and 10^{-5}.

Purification of VPMM. Purification of the virus proved to be quite difficult. In preliminary tests, the purification procedures devised by Reddy et al for PMV (12) and by R. H. Baum (*personal communication*) for bean yellow mosaic and clover yellow vein viruses resulted in no detectable virus. The procedure of Sun and Hebert (15) for a severe strain of PMV resulted in impure virus after two cycles of PEG precipitation but no virus band occurred after sucrose gradient centrifugation. A virus band was formed after cesium sulfate equilibrium centrifugation but the virus yield was low (0.2 mg virus/100 g infected *N. benthamiana* tissue).

Electron microscopy of VPMM. Purified virus particles had an average length of 773 nm (Fig. 4) and the rods were flexuous (Fig. 5) when stained with 2% ammonium molybdate.

In thin sections of infected *N. benthamiana* and *T. incarnatum* leaf tissues, cytoplasmic cylindrical inclusions composed of scrolls and pinwheels when viewed in cross section were observed (Fig. 6). This inclusion morphology places VPMM in inclusion body subdivision I of Edwardson (5).

Serology of VPMM. In gel-diffusion tests and ELISA, VPMM did not react with antisera to soybean mosaic, clover yellow vein, pea seedborne mosaic, watermelon mosaic I, potato Y, turnip mosaic, tobacco etch, and maize dwarf mosaic viruses. VPMM formed a faint precipitin line in gel-diffusion tests with antiserum to PMV, but BYMV had a stronger reaction with antiserum to PMV. In ELISA, VPMM also showed a slight reaction with antiserum to PMV (Table 1). In gel-diffusion tests, VPMM also had a slight reaction with antiserum to BYMV but PMV had a stronger reaction with antiserum to BYMV.

Molecular weight of coat protein subunit and RNA. Virus coat protein migrated as two bands in SDS-polyacrylamide gels. The molecular weight of the slower migrating polypeptide was 33,500. The faster migrating component had a molecular weight of 30,000 and was probably a breakdown product. VPMM RNA was a single component with a molecular weight of 3×10^6 after polyacrylamide gel electrophoresis.

Tests for resistance to VPMM. Of 663 peanut germ plasm lines tested, more than 80% of the plants in 646 lines were infected, 70–80% of 14 lines, and 50–70% of 3 lines. No peanut line with resistance to the virus was found.

DISCUSSION

On the basis of host range, transmission, in vitro properties, virus particle and inclusion body morphology, serology, physical and chemical properties, the virus causing an epidemic on peanuts in Hubei, China, was distinguished as a potyvirus that was different from previously reported potyviruses occurring on peanuts in nature. Because no antiserum has yet been produced to allow definitive serological tests and because of certain affinities to a virus reported by Moghal (10), it was considered premature to name the virus.

Peanut viruses are economically important in China. Yu (16), in 1939, presented results of a survey of peanut mosaic virus (PMosV) disease in Shandong. Chou and Cai (3), in 1959, reported that PMosV in Beijing infected

Table 1. Reaction ($o.d._{405}$) of virus causing peanut mild mottle (VPMM) with antiserum to peanut mottle virus (PMV) in ELISA

Experiment no.[a]	Antigens		
	PMMV	PMV	Healthy
1	0.65	1.84	0.47
2	1.14	0.95	0.48
3	0.45	1.60	0.305
4	0.29	1.69	0.23

[a] Experiments were done at different times with different *N. benthamiana* plants as sources of antigens. The reasons for the results of experiment 2 differing from those of the other experiments are not known.

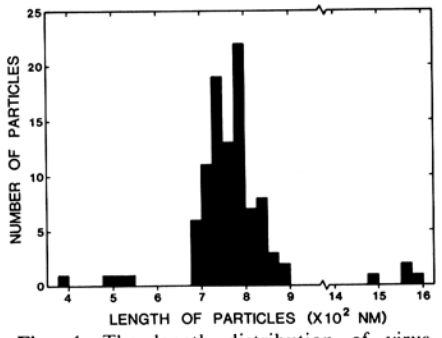
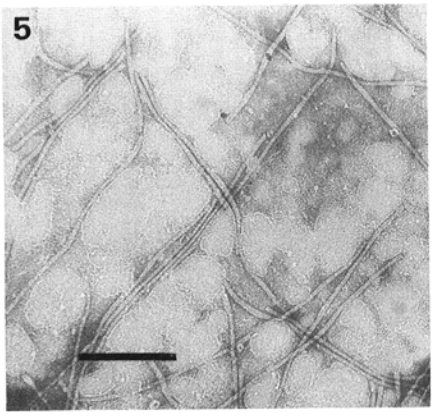
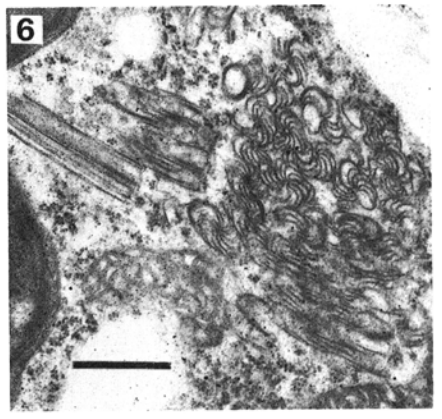


Fig. 4. The length distribution of virus particles causing peanut mild mottle.





Figs. 5 and 6. (5) Purified particles of the virus causing peanut mild mottle stained with 2% ammonium molybdate, pH 7.5. Scale bar = 240 nm. **(6)** Thin section of *Nicotiana benthamiana* infected with VPMM showed cytoplasmic cylindrical inclusions composed of scrolls and pinwheels. Scale bar = 1 μm.

peanut, *Dolichos lablab, Physalis floridana, Vigna unguiculata,* and *N. tabacum* among 13 species of plants tested by sap inoculation. Apparently, PMosV in Beijing has a wider host range than VPMM does. In 1979, Shih and Hsu (13) identified PMosV in Shandong. There are several differences between PMosV and VPMM. VPMM causes faint symptoms on peanut, whereas PMosV causes severe symptoms and stunting of infected peanut plants. VPMM has a comparatively narrow host range limited mainly to the leguminosae, whereas PMosV has a wide host range that includes some species of plants immune to VPMM, ie, *Phaseolus mungo, N. glutinosa, N. tabacum, Datura stramonium, Beta vulgaris,* and *Spinacia oleracea*. VPMM, however, can infect soybean cultivars that are immune to PMosV. VPMM has a thermal inactivation point of 55–60 C, which is higher than that of PMosV, 40–50 C. VPMM is seedborne but PMosV is not. Although VPMM and PMosV differ in several characteristics, both have flexuous particles of similar length.

Recently, another two potyviruses, groundnut eyespot virus (GEV) and peanut green mosaic virus (PGMV), were isolated from naturally infected peanut (4,14). VPMM differs from GEV and PGMV in symptomatology on peanut and on host range. GEV infects *Lycopersicon esculentum, Physalis floridana, Petunia hybrida,* and *B. vulgaris*, which VPMM does not infect. VPMM infects *C. amaranticolor* and *C. quinoa*, which GEV fails to infect. GEV is serologically closely related to soybean mosaic virus but VPMM did not react with soybean mosaic virus antiserum. Unlike VPMM, PGMV causes a severe mosaic and stunt on peanut. *Phaseolus vulgaris* is a good local host for PGMV. PGMV is not seedborne and is unrelated to PMV serologically.

Peanut mottle virus is the best characterized potyvirus naturally infecting peanut (2). Compared with PMV, VPMM does not cause depression of interveinal leaf tissue of infected peanuts, which is a characteristic of symptoms of PMV, although both VPMM and PMV cause a mild mottling on peanut (6). Although both have a host range limited mainly to leguminous plants, including peanut, soybean, *C. obtusifolia, C. occidentalis,* and *T. incarnatum*, VPMM and PMV both infect *N. clevelandii* and *N. benthamiana*, but VPMM infects *C. amaranticolor, C. quinoa,* and *C. album*, which are apparently immune to PMV (2,6). In contrast, PMV was reported to infect pea and bean in nature. Topcrop bean is an important local lesion host of PMV (2,6,7), which is not infected by VPMM. VPMM also does not infect some American soybean varieties, such as Bragg, Rampage, Bansei, and Davis, which are susceptible to PMV.

Moghal and Francki (11) and Edwardson (5) used the presence of laminated aggregates and scroll-type inclusions as a criterion for potyvirus identification and classification. VPMM has scroll-type pinwheels, which are placed in inclusion body subdivision I of Edwardson (5), whereas PMV, which has both laminated aggregates and scroll-type inclusions, is placed in subdivision III. Because of these several differences between VPMM and PMV, it seems certain that VPMM is not PMV even though they are related serologically.

A virus with many of the properties of PMV was described by Behncken in Australia (1). The host range of the Australian PMV was similar to that reported by Kuhn (6) for PMV, and on the basis of host range and other properties, the Australian PMV was thought to have been imported from the United States. Moghal (9) studied a PMV isolate obtained from Behncken that had a host range very similar to that reported by Behncken (1) but had an inclusion body morphology like VPMM (ie, no laminated aggregates such as occur in PMV inclusions). The relationship between Australian PMV and the VPMM needs to be examined and the serological relationships of both to PMV established with sera to all three viruses.

VPMM causes faint symptoms on peanut and is frequently overlooked; however, because VPMM causes a 23% yield loss of peanut and a 53% yield loss of soybean and is prevalent in most peanut-growing areas, control measures for the virus should be sought and implemented.

ACKNOWLEDGMENTS

We thank Joan Hudson for electron microscopy and M. T. Zimmerman and R. E. Baker for technical assistance. We thank Duan Naixiong for his cooperation in the tests of peanut germ plasm lines for resistance to the virus.

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