Seed coats may become unevenly thickened. All stages of the nematode are found throughout the seed coat until the end of the growing season, when mainly juvenile stages are found. *A. arachidis* predisposes seed to invasion by soilborne fungal pathogens such as *Rhizoctonia solani, Sclerotium rolfsii, Macrophomina phaseolina,* and *Fusarium* spp., leading to reduced seedling emergence. *A. arachidis* devalues the crop by reducing seed quality.

A. arachidis can survive desiccation in stored pods for up to 12 months, while infected pods that are sun dried in the field before storage contain no active nematodes. Adult nematodes have been isolated from volunteer plants. Control of this nematode is mainly by preventive treatments that include immersing seed in 60°C water and sun drying pods after harvest. *A. arachidis* can be disseminated in infected seed and therefore has the potential to become a widely distributed pest.

Aphasmatylenchus structuratus Germani is a migratory endoparasite and ectoparasite of peanut that has been reported only in southwest Burkina Faso in western Africa. The nematode causes chlorosis, stunting, reduced development of the root system and *Rhizobium* nodules, and yield reduction. *A. structuratus* survives the dry season adjacent to roots of the karite tree (*Butyrospermum parkii* L.) and does not enter into anhydrobiosis. This nematode spreads rapidly and parasitizes other economically important leguminous plants in Burkina Faso.

Scutellonema cavenessi Sher has been found in northern Nigeria, Senegal, and Mali. This nematode causes chlorosis, reduced root growth, and reduced *Rhizobium* nodulation. *S. cavenessi* is active during the rainy season and enters into anhydrobiosis when soil moisture drops to approximately 0.2%. The extent of yield loss from this nematode is not known, but the application of nematicides increases yield 20–220%. *S. cavenessi* has been found associated with most cultivated plants in Senegal and Mali. Leaving fields fallow between crops provides excellent control but is not economically practical.

Tylenchorhynchus brevilineatus Williams (syn. *T. indicus* Siddiqi) has been reported only in the Kalahasti area and Nellore district of Andhra Paradesh, India. This nematode causes a brownish black discoloration of the pod surface and reduced pod size. Pegs and young pods may have brownish yellow lesions with slightly raised margins. *T. brevilineatus* can be controlled and yields increased with application of aldicarb or carbofuran.

Dagger nematodes (*Xiphinema* spp.) are found consistently in peanut and often damage roots, producing galls and curly tips. Populations of dagger nematodes in peanut fields are typically very low and stable throughout the growing season, making the damage they cause relatively unimportant.

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(Prepared by N. Kokalis-Burelle and R. Rodríguez-Kábana)

Diseases Caused by Viruses

Tomato Spotted Wilt and Peanut Bud Necrosis

The peanut bud necrosis virus (PBNV) and tomato spotted wilt virus (TSWV), both tospoviruses, cause economically important diseases in peanut. The distinction between PBNV and TSWV has been recognized only recently. The disease referred to as "bud necrosis," now shown to be caused by either TSWV or PBNV, was previously attributed only to TSWV. TSWV and PBNV cannot be distinguished by symptoms alone on peanut or other hosts. TSWV is widely distributed in the Americas, Australia, Africa, and Europe, whereas PBNV appears to be restricted to southern and southeastern Asia. Nevertheless, the thrips vectors of TSWV and PBNV occur in most peanut-growing countries. Therefore, in surveys for the occurrence of virus diseases in peanut, assays for both PBNV and TSWV should be conducted.

Symptoms

Symptoms caused by TSWV and PBNV in peanut are variable. They may appear in young leaflets in the form of chlorotic spots or a mild mottle (Plate 99) that develops into necrotic and chlorotic rings and streaks. Bud necrosis symptoms appear to be governed by ambient temperatures; when temperatures are above 30°C during the day, petioles bearing fully expanded leaflets with initial symptoms (as described above) usually become flaccid and droop (Plate 100). This symptom is followed by necrosis of the terminal bud. When a relatively young plant is infected, the necrosis spreads toward the base of the plant, resulting in its death. Secondary symptoms include stunting and proliferation of axillary shoots. Leaflets produced on the axillary shoots are reduced in size and show puckering, mosaic mottling, and general chlorosis (Plate 101). Secondary symptoms are most common on early-infected plants, giving them a stunted and bushy appearance. Plants infected later may also be stunted, but the symptoms may be restricted to a few branches or to the apical parts of the plants. Seed from early-infected plants are small and shriveled, and the seed coats are red, brown, or purple with mottling (Plate 102). Although late-infected plants may produce seed of normal size, the seed coats are often mottled and cracked.

Causal Agents

TSWV and PBNV are found in all parts of affected plants. Clusters of virus particles are often found in the cisternae of the endoplasmic reticulum. Individual particles are 80–120 nm in diameter and are covered with projections resembling spikes (Fig. 71). TSWV and PBNV have an extremely low thermal inactivation point (45°C for 10 min) and short longevity in vitro (less than 5 hr at room temperature). These properties can be used, in conjunction with others, to identify TSWV and PBNV.

It is difficult to isolate TSWV or PBNV from infected plant tissues. Additionally, neither TSWV nor PBNV is highly immunogenic. Polyclonal antibodies produced for an isolated virus may react with healthy plant extracts. Therefore, extreme care should be taken in using polyclonal antisera and in interpreting the results of serological tests. Polyclonal or mono-

clonal antibodies are widely used to study relationships among tospoviruses and to diagnose disease.

Host Ranges

TSWV and PBNV have extremely wide host ranges that include more than 370 species of plants in more than 50 families.

Transmission

Both TSWV and PBNV are mechanically transmissible. Only chilled extracts containing antioxidants such as mercaptoethanol or thioglycerol are suitable for transmitting the virus by mechanical inoculations. Both TSWV and PBNV are transmitted by thrips. Probable vectors of TSWV in the United States are *Frankliniella fusca* (Hinds) and *F. occidentalis* (Pergande); *Thrips palmi* (Karny) transmits PBNV in India. The viruses are acquired only by insect larvae but may be transmitted by larvae or adults. TSWV multiplies in its thrips vector. Neither PBNV nor TSWV is transmitted by seed in peanut.

Control

Cultural practices such as early seeding, use of high-quality seed treated with an approved seed protectant, and sowing at the recommended rate and spacing to give optimum plant population can reduce the incidence of TSWV and PBNV. The seedbed should be well prepared, and soil moisture should be sufficient to ensure good germination and seedling establishment. Given good growing conditions, the crop will rapidly develop a close canopy and will not be as attractive to the thrips vector as a patchy crop. The incidence of TSWV and PBNV under these conditions is much reduced. Because of the extremely wide host ranges of TSWV and PBNV and their vectors, it is not practical to control the disease by destroying weed reservoir hosts. Intercropping one row of a quick-growing cereal crop such as sorghum or pearl millet with each three rows of peanut can reduce disease incidence. Removal of infected plants will create gaps in the field that may lead to an increase in the percentage of infected plants.

Good sources of resistance to both TSWV and PBNV have been identified. The cultivar Southern Runner typically has 50% lower disease incidence than the susceptible cultivar Florunner. Peanut genotypes resistant to PBNV and *T. palmi* include ICGV 86029, ICGV 86031, and ICGV 86388. Southern Runner is also resistant to PBNV. Since the application of insecticides increases disease incidence of both PBNV and TSWV, it is not advisable to apply insecticides for control.

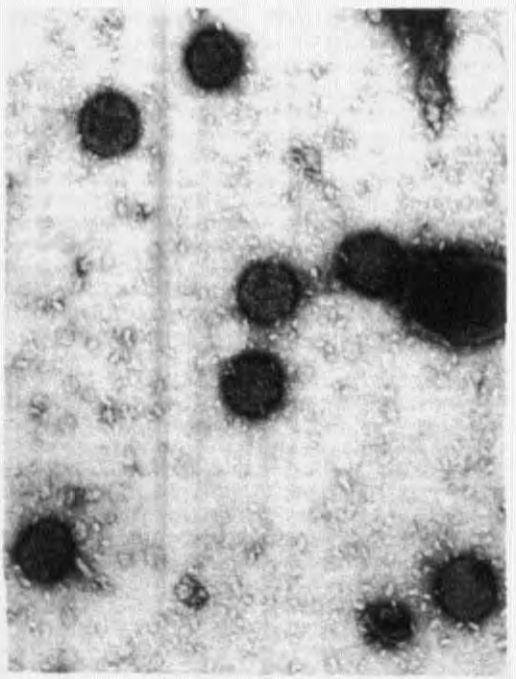

Fig. 71. Particles of peanut bud necrosis virus covered with spikes or projections.

Peanut Clump and Indian Peanut Clump

Peanut clump occurs in the Indian subcontinent and in western Africa (Senegambia, Burkina Faso, Niger, and the Ivory Coast). The pathogen, a soilborne virus, causes severe crop losses. Symptoms of peanut clump resemble those of green rosette (see Groundnut Rosette). As a result, it is likely that these two diseases have been confused.

Studies on genome organization have revealed that isolates from India and western Africa are two distinct viruses. The virus from western Africa, referred to as peanut clump virus (PCV), and that from India, Indian peanut clump virus (IPCV), are not serologically related. Differences in genome organization between PCV and IPCV are apparent, and the complete nucleotide sequences of both genome RNAs are available.

Symptoms

Diseased plants are severely stunted and dark green (Plate 103). The disease occurs in patches in the field and recurs more or less in the same position in the same field in successive peanut crops. The symptoms first appear on young plants as mottling, mosaic, and chlorotic rings on newly emerged quadrifoliates (Plate 104). Subsequently, infected leaves turn dark green with or without faint mottling. Early-infected plants are severely stunted and may produce flowers, but any pods formed are not well developed. Plants infected later are also stunted and have shortened internodes and dark green leaflets. These plants may produce pods, but seed weights may be reduced by up to 60%.

Causal Agents

PCV and IPCV have rod-shaped particles 24 nm in diameter; there are two predominant lengths, approximately 185 and 250 nm (Fig. 72). The particles of PCV and IPCV each contain a single polypeptide species with a molecular mass of 24 kDa.

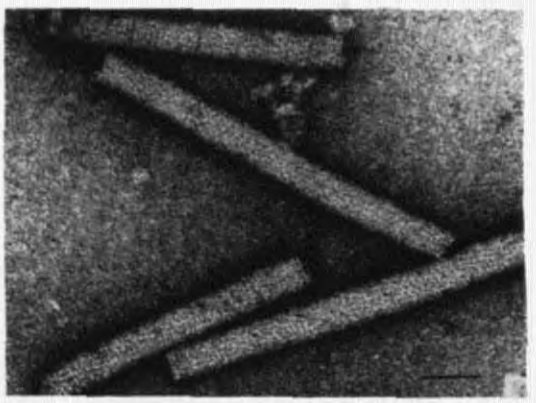

Fig. 72. Peanut clump virus particles.

Both IPCV and PCV are currently known to occur in several serologically distinct variants. Among the IPCV variants, three distinct serogroups have been identified. Variants can also be distinguished on the basis of their reaction on several hosts. RNA1 of IPCV has been found to contain sequences conserved among the variants and will be utilized to produce nucleic acid probes capable of detecting several IPCV variants.

Host Ranges

PCV and IPCV have extremely wide host ranges, which include many monocotyledonous and dicotyledonous plants.

Transmission

IPCV and PCV are readily sap transmissible. Both are transmitted by *Polymyxa graminis*. Both viruses are transmitted by seed in peanut (more than 6% frequency). Since IPCV is present in seed coats of all seed from infected plants, the seed coats should be removed before ascertaining seed transmission. IPCV is seed transmitted in cereal crops such as finger millet, foxtail millet, and pearl millet but not in sorghum.

The occurrence of IPCV is correlated with ambient air temperatures. When ambient air temperatures are below 25°C, only negligible IPCV incidence is observed. Therefore, in the tropics, crops grown after the rainy season in locations where temperatures are lower than 25°C escape infection. High temperatures during the summer season followed by monsoon rains appear to favor natural transmission.

Control

IPCV incidence is higher when peanut crops are rotated with susceptible cereal crops. The incidence of virus can be reduced by the application of soil biocides. Soil solarization (during hot summer months for a period of 2 months) reduces IPCV incidence. None of more than 9,000 *Arachis hypogaea* genotypes showed resistance to IPCV when tested on infested soils. Since the genomes of PCV and IPCV have been sequenced, there are excellent prospects for utilizing viral genes to induce resistance by unconventional methods in *A. hypogaea*.

Groundnut Rosette

Groundnut rosette disease was first reported from Tanzania in 1907. Many epidemics of rosette were subsequently recorded in Africa. One major epidemic in 1975 caused nearly $250 million in crop losses in Nigeria alone.

Rosette has also been reported in Argentina, India, Indonesia, and the Philippines. In Africa, rosette disease is restricted to countries south of the Sahara.

Symptoms

Three types of rosette disease, chlorotic, mosaic, and green, are recognized on the basis of symptoms. Chlorotic rosette occurs throughout Africa south of the Sahara. The disease first appears on young leaflets as faint mottling with a few green islands. Leaflets produced subsequently are pale yellow with green veins. Plants infected when young produce progressively smaller, chlorotic, curled, and distorted leaflets. When older plants are infected, symptoms may be restricted to a few branches or to the apical portion of the plants. Plants infected early are severely stunted (Plate 105) with thickened stems. Early infection causes severe reduction in the number and size of pods.

Green rosette disease occurs in western Africa and Uganda. Young leaflets show mild chlorotic mottling and isolated flecks. Symptoms are masked in older leaflets, but leaflets are reduced in size, show outward rolling, and are not distorted. Plants infected early are severely stunted and are a darker green than healthy plants (Plate 106), somewhat resembling plants infected with peanut clump virus.

Mosaic rosette occurs only in eastern and central Africa. Young leaflets show conspicuous mosaic symptoms (Plate 107), which resemble those of chlorotic rosette except that stunting is less pronounced.

Causal Agents

Rosette disease of peanut is caused by a complex of two viruses and a satellite RNA. Diseased plants contain a mechanically transmissible virus, groundnut rosette virus (GRV), which is classified as an umbravirus. GRV depends on the groundnut rosette assistor virus (GRAV), a luteovirus, for transmission by *Aphis craccivora*. GRAV causes no obvious symptoms in peanut on its own. Variants of the satellite RNA are responsible for the different forms of rosette (chlorotic, green, and mosaic). These variants differ appreciably in nucleotide sequence, as do forms of the satellite from different parts of Africa.

GRAV, a typical luteovirus, can be detected by polyclonal antisera produced against GRAV and by antisera to some other luteoviruses, such as potato leafroll virus (PLRV). It can also be detected by some monoclonal antibodies to PLRV. No virus particles have been reported for GRV, and so no antiserum is available. Infected plants contain abundant infective single-stranded RNA.

Host Range

Peanut is the only natural host known for both GRAV and GRV, though alternate hosts probably play an important part in perpetuating the inoculum between crop seasons. Most species become infected with only one virus or the other. Host range tests with GRV can be done also by mechanical inoculation. GRV cultures, with or without the satellite RNA, induce necrotic lesions in host plants but not systemic infections. Necrotic rings are also produced on inoculated leaves. Most sensitive test plants produce mild veinal chlorosis or necrosis on the first systemically infected leaves, which is followed by a faint mottle and moderate stunting.

Transmission

The viruses associated with all three types of rosette disease are transmitted by *A. craccivora* in a persistent or circulative manner. By analogy with other umbraviruses, it is suspected that GRV and its satellite RNA are encapsidated in GRAV coat protein. Peanut is considered to be the main source of inoculum from which the initial spread of the rosette disease occurs. Neither GRAV nor GRV is seed transmitted in peanut. Because rosette-infected plants survive longer than healthy plants, they are not normally harvested and serve as an important source of inoculum. Volunteer plants may also be a source of inoculum. It is likely that *A. craccivora* colonizes these rosette-infected plants and that moving rainy fronts are responsible for the dissemination of the aphids. Alate and apterous aphids are involved in the secondary spread. Diagnostic aids for the various components of rosette have recently become available and should permit epidemiological studies on rosette disease in Africa.

Control

Rosette disease can be effectively controlled by cultural practices. These include destroying all volunteer and unharvested infected plants, planting early in the season and at a high seeding rate, maintaining plant stands, and applying insecticides at the correct time. Excellent sources of resistance to rosette disease are available in peanut germ plasm. Early-maturing, rosette-resistant genotypes recently have been identified and are being used in the development of rosette-resistant, early-maturing cultivars. Since the coat protein gene of GRAV has been sequenced and constructs suitable for transformation have been prepared, prospects for producing rosette-resistant cultivars by insertion of the coat protein genes into peanut are good.

Peanut Mottle

Peanut mottle virus (PeMoV) was first reported in the United States in 1961 and is currently present in all the major peanut-growing countries. Its widespread distribution is probably the result of dispersal of infected seed. An economic loss of 5–6% from PeMoV was estimated in Georgia. In field tests in India, susceptible cultivars suffered yield losses of up to 40%. Because of its worldwide distribution and potential for causing economic losses, PeMoV is considered to be of global economic importance.

Symptoms

Young leaflets show mild mottle symptoms or a mosaic of irregular, dark green islands (Plate 108). In older leaflets, mosaic symptoms are not obvious but can be seen by transmitted light. In some genotypes, conspicuous interveinal depressions and an inward curling of the edges of leaflets are apparent (Plate 109). Infected plants are slightly stunted. PeMoV reduces both numbers and size of pods on infected plants.

Causal Agent

PeMoV belongs to the potyvirus group. Particles are flexuous rods that measure about 750 ×12 nm (Fig. 73). The coat protein has an apparent molecular mass of 32–36 kDa. The thermal inactivation point is 55–64°C, and the longevity in vitro is 1–2 days at room temperature.

High-quality polyclonal antisera and monoclonal antisera have been produced for PeMoV. They do not react with the peanut stripe virus or the peanut green mosaic or groundnut eyespot potyviruses that occur on peanut.

Host Range

PeMoV occurs in several important legume crops, including peanut and soybean, and weeds.

Transmission

PeMoV is readily transmitted by mechanical sap inoculation and is seed transmitted at rates of 0–8.5%. PeMoV is also seed transmitted in mung bean and cowpea but not in soybean. The virus can be detected in peanut seed by enzyme-linked immunosorbent assay. PeMoV is transmitted in a nonpersistent manner by *Aphis craccivora*, *A. gossypii*, *Myzus persicae*, *Hyperomyzus lactucae*, *Rhopalosiphum padi*, and *R. maidis*.

Control

Peanut seed appears to be the primary source of inoculum. Since many aphid species can transmit the virus, it is spread rapidly to nearby plants. To avoid the disease, the planting of virus-free seed is important. Genotypes in which PeMoV is not seed transmitted have been identified and utilized in conventional breeding programs to produce acceptable, high-yield breeding lines that do not transmit the disease via seed. Although resistance to PeMoV has been identified in wild species of *Arachis*, it has not yet been transferred to *A. hypogaea*.

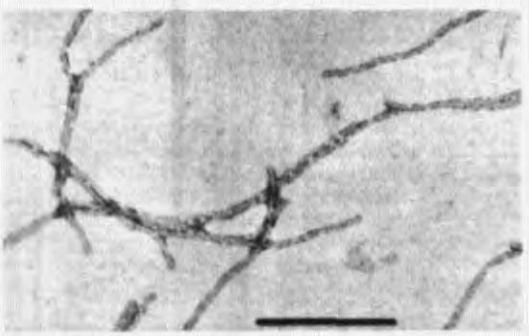

Fig. 73. Peanut mottle virus particles. Bar = 500 nm. (Courtesy C. Kuhn)

Peanut Stripe

The peanut stripe virus (PStV) was first reported in the United States in 1983, having entered the country in peanut seed imported from China. PStV has been present in Southeast Asia since the early 1970s but has been often misidentified as peanut mottle virus (PeMoV). Peanut stripe poses a serious threat to peanut production in southern and southeastern Asia.

Symptoms

Several symptom variants of PStV are known. The name peanut stripe virus was first given to an isolate that induced discontinuous, dark green stripes along the lateral veins of young leaflets (Plate 110). However, the most widely distributed variant causes irregular green blotches on young leaflets that persist as the leaflets age (Plate 111). A variant that induces chlorotic rings surrounding blotches on young leaflets was reported from Thailand and Indonesia. The most widely distributed isolate in China induces a mild mottle symptom (Plate 112).

Causal Agent

PStV belongs to the potyvirus group. Although its particles resemble those of PeMoV, PStV is serologically distinct from PeMoV. However, comparison of available full-length potyvirus nucleic acid sequences indicates that PStV is closely related to the soybean mosaic virus.

Host Range

Natural hosts in the field are *Centrosema pubescens*, *C. macrocarpum*, *Calopogonium caeruleum*, *Crotalaria striata*, *Desmodium siliquosum*, and *Pueraria phaseoloides*. A number of hosts can be systemically infected with PStV by sap inoculation. Since PStV does not produce local lesions on the *Phaseolus vulgaris* cultivar Topcrop, this host can be used to distinguish it from PeMoV.

Transmission

PStV is transmitted by sap inoculation and by many aphid species in a nonpersistent manner. Different symptom variants of PStV have different aphid transmission frequencies. Seed transmission of PStV can be as high as 37% when the seed are derived from plants inoculated before flowering. Seed transmission frequency in naturally infected plants is normally less than 5%. The virus is readily detected in seed by enzyme-linked immunosorbent assay.

Control

Of approximately 10,000 peanut genotypes evaluated for resistance to PStV in Indonesia, none was resistant. However, some genotypes showed only mild symptoms, and some took a longer time than the susceptible check to show overt symptoms. The PStV genome has been sequenced, and the potential exists for utilizing viral coat protein genes to incorporate resistance into *Arachis hypogaea*.

In areas where PStV is established, it occurs at high incidence, resulting in the harvest of virus-contaminated seed. The common practice of using seed from the previous season's crop assures the continuous presence of PStV in the field. Therefore, production and distribution of virus-free seed should be given a high priority in efforts to contain the spread of PStV.

Peanut Stunt

Peanut stunt was first observed in the United States in 1964. It was economically important in the southeastern United States in various forage legumes and beans, but it is now only a minor disease. The peanut stunt virus (PSV) occurs naturally in peanuts from Sudan and China, where it can cause crop losses of up to 75%.

Symptoms

In the United States, PSV causes severe dwarfing of the entire plant or of one or more branches. In China, the virus does not cause severe stunting. Shortening of the petioles, reduction in the size of leaflets, chlorosis, and malformation are observed in the United States, China, and Sudan (Plate 113). Plants infected early in the growing season produce very few pods, and these are misshapen and frequently have a split pericarp wall. The viability of seed from such pods is markedly reduced. The virus causes epinasty with systemic mosaic and malformation in cowpea (cultivar Blackeye). Systemic symptoms produced by PSV in peanut, beans, and cowpea can be used to distinguish it from the cucumber mosaic virus.

Causal Agent

PSV belongs to the cucumovirus group. The particles are 25–30 nm in diameter and encapsidate three single-stranded RNAs. Two serologically distinct isolates from the United States, PSV-E from the eastern region and PSV-W from the western region, and three serotypes from China, PSV-T, PSV-2, and PSV-B2, have been reported.

Host Range

PSV has a wide host range. It produces local lesions on *Chenopodium amaranticolor* and *C. quinoa*.

Transmission

PSV is transmitted by sap inoculation and in a nonpersistent manner by three aphid species, *Aphis craccivora*, *A. spiraecola*, and *Myzus persicae*. It is seed transmitted at the lowest frequency of all the other known seed-transmitted peanut viruses. Up to 0.01% of large seed from plants infected late in the season may contain the virus. Up to 0.2% of small seed from less severely stunted plants may be infected.

Control

Since some forage legumes, such as white clover, are the primary source of inoculum, peanuts should not be planted in fields located near such legumes. Roguing of infected plants from crops intended for seed production is recommended. Currently, there are no peanut genotypes resistant to PSV.

Cowpea Mild Mottle

The cowpea mild mottle virus (CPMMV) is widely distributed in Asia and Africa but has not been reported in the United States. CPMMV incidence in peanut does not exceed 5%. Because of its wide distribution, potential to cause severe crop losses, and occurrence at high incidence in peanut crops intercropped or grown adjacent to crops such as soybean or cowpea, incidence of CPMMV should be routinely monitored in countries where it is endemic.

Symptoms

Initial symptoms on young leaflets are veinclearing followed by downward rolling of the leaflet edges and veinbanding. Subsequently, necrosis of leaflets and petioles occurs. Plants are severely stunted and are conspicuous because of the rolled edges and veinbanding of the leaflets (Plate 114).

Causal Agent

CPMMV is a member of the carlavirus group of plant viruses. The particles are slightly flexuous rods, 15 nm in diameter and 610 nm long (Fig. 74). The thermal inactivation point of CPMMV is 75–80°C. High-quality polyclonal antisera to CPMMV also react with the groundnut crinkle virus reported from the Ivory Coast. CPMMV may be serologically related to several aphid-transmitted carlaviruses.

Host Range

The virus produces local lesions and systemic symptoms on many hosts.

Transmission

CPMMV is readily sap transmissible. The whitefly, *Bemisia tabaci*, transmits the virus in a nonpersistent manner. CPMMV is not seed transmitted in peanut.

Control

Peanut crops should not be planted adjacent to crops such as cowpea and soybean, which are frequently colonized by *B. tabaci* and are highly susceptible to infection by CPMMV in areas where it occurs.

Cucumber Mosaic

Natural occurrence of the cucumber mosaic virus (CMV) in peanut has been reported only from China. The disease caused by CMV is referred to as peanut yellow mosaic and is currently recognized as economically important in the northern regions of China. CMV has caused crop losses of up to 40%.

Symptoms

Initial symptoms are chlorotic spots and upward rolling of young leaflets. Subsequently produced leaflets show a yellowing of the lamina with green stripes along the lateral veins (Plate 115). Occasionally, leaflets are deformed and plants are moderately stunted. The severe yellowing and mottling symptoms observed on young plants are not apparent on older plants.

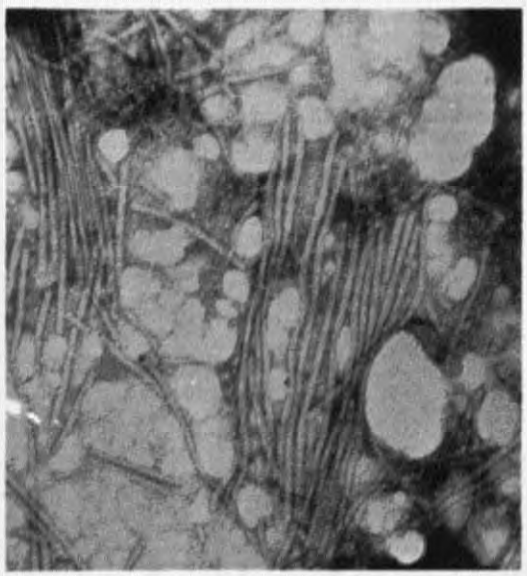

Fig. 74. Cowpea mild mottle virus particles.

Causal Agent

Virus particles are 29 nm in diameter. Many strains of CMV occur in crop plants. Two strains of CMV naturally infect peanut in China. A strain of minor importance, CMV-CS, is serologically related to the peanut stunt virus. The predominant strain, CMV-CA, is serologically related to CMV-D but is distinct from CMV-CS.

Host Range

CMV strains have wide host ranges. CMV-CA can infect 31 plant species in six families by sap inoculation.

Transmission

CMV-CA is easily transmitted by sap inoculation and by several aphids. Seed transmission is up to 4% in peanut.

Control

Seed from infected crops should not be planted. Cultural measures such as mulching with transparent plastic sheets and roguing out diseased seedlings at early stages of crop growth can reduce disease incidence. No resistance to CMV has been identified in the cultivated peanut.

Peanut Chlorotic Streak

Peanut chlorotic streak was first observed in 1977 in Andhra Pradesh, India. In subsequent surveys, the causal agent, the peanut chlorotic streak virus (PCISV), was found to be widely distributed in India. It was reported to be a new member of the caulimovirus group. Recently, a symptom variant of PCISV, which is referred to as the "veinbanding isolate" and has minor differences in the physical map of the genome, has also been reported.

Symptoms

Characteristic symptoms appear on young peanut leaflets as oval, chlorotic streaks along the veins. Leaflets are reduced in size, and early-infected plants are stunted. Symptoms are not distinct on older leaflets.

Causal Agent

Purified virus particles of PCISV are 52 nm in diameter. Purified virus contains two polypeptides with molecular masses of 58 and 51 kDa. PCISV DNA has recently been fully sequenced, and the majority of the gene products indicate significant relationships to other caulimoviruses. In enzyme-linked immunosorbent assays, PCISV does not react with cauliflower mosaic, figwort mosaic, or soybean chlorotic mottle viruses.

Host Range

The host range of PCISV is unusually diverse compared with other caulimoviruses. Chlorotic lesions with necrotic centers produced on cowpea are very characteristic. Systemic infection occurs on several *Nicotiana* species, *Datura stramonium*, *Glycine max*, *Petunia* × *hybrida*, *Spinacea oleracea*, and *Vigna radiata*.

Transmission

PCISV is readily mechanically transmissible. It is not transmitted by *Aphis craccivora*, *Myzus persicae*, or *Bemisia tabaci* and is not seed transmitted.

Control

The field incidence of PCISV does not exceed 1%. However, the veinbanding variant of PCISV was observed at an incidence exceeding 20%. No control measures are currently available.

TABLE 6. Viruses That Naturally Infect Peanut[a]

Name	Taxonomic Group	Family	Distribution
Cowpea chlorotic mottle virus	Bromovirus	Bromoviridae	United States
Cowpea mild mottle virus	Carlavirus	NA[b]	China, India, Indonesia, Ivory Coast, Nigeria, Thailand, Philippines, Papua New Guinea, Sudan
Groundnut crinkle virus	Carlavirus	NA	Ivory Coast
Peanut chlorotic streak virus	Caulimovirus	Pararetroviridae	India
Peanut chlorotic streak virus (veinbanding isolate)	Caulimovirus	Pararetroviridae	India
Cucumber mosaic virus	Cucumovirus	Bromoviridae	China
Peanut stunt virus	Cucumovirus	Bromoviridae	Sudan, Japan, Spain, United States
Peanut clump virus	Furovirus	NA	Niger, Burkina Faso, Ivory Coast, Senegal
Indian peanut clump virus	Furovirus	NA	India, Pakistan
Groundnut yellow mosaic virus (bean golden yellow mosaic virus)	Geminivirus	Geminiviridae	India
Tobacco streak virus	Ilarvirus	NA	Brazil
Groundnut rosette assistor virus	Luteovirus	NA	All of Africa south of the Sahara
Sunflower yellow blotch virus	Luteovirus	NA	Malawi, Kenya, Zambia, Tanzania
Groundnut veinal chlorosis virus	Rhabdovirus	Rhabdoviridae	India, Indonesia
Groundnut chlorotic spotting virus	Potexvirus	NA	Ivory Coast
Bean yellow mosaic virus	Potyvirus	Potyviridae	United States
Groundnut eyespot virus	Potyvirus	Potyviridae	Ivory Coast
Passion fruit woodiness virus	Potyvirus	Potyviridae	Australia
Peanut green mosaic virus	Potyvirus	Potyviridae	India
Peanut mottle virus	Potyvirus	Potyviridae	Worldwide
Peanut stripe virus	Potyvirus	Potyviridae	Brazil, China, India, Indonesia, Japan, Malaysia, Philippines, Myanmar, Thailand, Taiwan, Vietnam, United States
Peanut bud necrosis virus	Tospovirus	Bunyaviridae	India, Nepal, Sri Lanka, China, Taiwan, Indonesia, Thailand
Peanut yellow spot virus	Tospovirus	Bunyaviridae	India, Thailand
Tomato spotted wilt virus	Tospovirus	Bunyaviridae	North America, South America, South Africa, Nigeria
Groundnut yellow mottle virus	Tymovirus	NA	Nigeria
Groundnut rosette virus	Umbravirus	NA	All of Africa south of the Sahara

[a] Listed alphabetically by taxonomic group.
[b] Not yet assigned.

Other Viruses

Many other viruses, including those that cause groundnut streak mosaic (Plate 116), groundnut crinkle, groundnut eyespot, peanut green mosaic, peanut yellow spot (Plate 117), peanut yellow mottle, groundnut streak, marginal chlorosis, and rugose leaf curl, have been associated with peanuts throughout the world. Although these pathogens often cause sporadic infections, yield losses can be significant.

Known viruses that can infect peanut under natural conditions, their geographical distributions, and the taxonomic groups to which they belong are listed in Table 6.

Witches'-Broom

Witches'-broom is caused by a phytoplasma (formerly called a mycoplasmalike organism). Unlike viruses, phytoplasmas are cellular organisms related to bacteria. The disease is characterized by stunting and excessive proliferation of shoots from axils (Plate 118). Plants are bushy in appearance. Leaflets are pale yellow and small. Pegs tend to grow upward, and pod yields are severely reduced.

A high incidence of witches'-broom on peanut has been observed in parts of Taiwan, Indonesia, and the Philippines. The pathogen is also known to occur in India, Thailand, China, and Papua New Guinea. A polyclonal antiserum has been produced for the detection of phytoplasma by use of enzyme-linked immunosorbent assay and can be used to distinguish witches'-broom from viral diseases characterized by severe stunting and a bushy appearance.

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