CHAPTER 12

# VIRUS DISEASES*

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EARLIER reviews written from India on groundnut virus diseases dealt with viruses occurring in other countries also (Raychaudhury and Nariani, 1977; Narayanasamy, 1983). This review will exclusively deal with various aspects of groundnut virus diseases in India which include symptoms, details of causal virus, methods for diagnosis and various ways of managing groundnut virus diseases.

Several virus diseases of groundnut have been reported from India. Reports prior to 1976 were based upon the symptoms, host range and transmission. No attempts were made to characterize the causal viruses. Inter-relationships with similar viruses occurring in other countries have not been investigated. It is well known that external symptoms by viruses can be greatly influenced by genotype, plant age, environment and strain of virus present. Recently Hamilton *et al.* (1981) outlined the various basic procedures which should be used for the identification and characterization of plant viruses. One of the most recent reviews on groundnut viruses (Narayanasamy, 1983) listed all the viruses reported so far from India without any discussion on the validity of the various reports, thus maintaining the confusion that currently exists. Since several groundnut viruses occurring in India have been characterized, I have taken the liberty of listing the probable causal viruses for majority of viral diseases of groundnut in India. A detailed account on viruses which are thoroughly characterized has been given here.

For the last seven years ICRISAT staff have conducted extensive surveys in India in all the major groundnut-growing areas. With the exception of minor viruses or those restricted to locations in India not covered by us in our disease surveys, all the major groundnut viruses have been characterized at the ICRISAT, antisera have been produced and inter-relationships with similar viruses occurring in other countries have been determined. Currently we are investigating various methods to manage groundnut virus diseases.

## BUD NECROSIS DISEASE (BND)

On the basis of symptoms, it appears that bud necrosis in India, which was first reported by Reddy *et al.* (1968), has been described under 7 different names: groundnut mosaic (Nariani and Dhingra, 1963), groundnut rosette (Bisht *et al.*, 1963), bunchy top, chlorosis, ring mottle (Sharma, 1966), bud blight (Chohan, 1972) and ring mosaic (Narayanasamy *et al.*, 1975). The disease is widely distributed in India. It occurs in high incidence in parts of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra and Uttar Pradesh. BND is currently regarded as the most economically important virus disease in India. Accurate estimates of yield loss due to BND have not been reported so far. In early infections it can cause up to 100% loss in yield (Prasada Rao *et al.*, 1979). Yield losses depend on the age of the plant, growth habit, cultivar,

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various factors in soil and environmental factors, and compensation, if any, to yield was derived by healthy plants adjacent to infected plants (Amin and Reddy, 1983).

### Symptoms

A wide variety of symptoms have been recorded which appear to be the main reason for the description of BND under different names by different authors. Initial symptoms under








**Fig. 12.1.** *1* Chlorotic spots induced by tomato spotted wilt virus *2* Chlorotic and necrotic rings induced by tomato spotted wilt virus, *3* Axillary shoot proliferation, severe leaf-deformity and stunting induced by tomato spotted wilt virus; *4* Shrivelled and mottled kernels from early infected plants *(right)*, kernels from uninfected plants *(left)*, *5* Electron micrograph of a thin section of leaflet infected by tomato spotted wilt virus (bar represents 240 nm), *6* Purified virus particles of tomato spotted wilt virus (bar represents 155 nm)

field conditions appear in about 40 days after germination on young leaflets as chlorotic spots with few necrotic spots or concentric chlorotic rings or chlorosis with green islands (Fig. 12.1.*1,2*). The fully-expanded quadrifoliates immediately below the terminal bud often become flaccid. The necrosis of the terminal bud follows and it appears to be more common in summer crops indicating that the symptom is probably influenced by high temperatures. If the bud necrosis occurs on the young plants, it may spread to other parts leading to death of the plant. On the older plants the necrosis usually spreads to the petiole and to a portion of the stem immediately below the necrosed terminal bud. This symptom is followed by stunting and axillary shoot proliferation. Leaves formed on the axillary shoots show a wide range of symptoms including reduction in size, distortion of lamina, mosaic mottling and general chlorosis (Fig. 12.1.*3*). Sometimes the lamina is reduced to midrib giving the leaflet a shoe-string appearance. Thus early infected plants are stunted and bushy and could be regarded as infected with 'rosette' disease. However, symptoms of both chlorotic and green rosette are totally different from BND symptoms (Table 12.1). Symptoms of green rosette can hardly be regarded as due to BND. Seeds from early infected plants are mottled and shrivelled (Fig. 12.1.*4*).

**Table 12.1.** Differences between bud necrosis disease and chlorotic rosette[1]

| Characteristics | Bud necrosis disease | Chlorotic rosette |
|---|---|---|
| Primary symptoms | Young quadrifoliates show chlorotic spots with mottling. Terminal bud necrosis may follow the initial symptom. Concentric chlorotic rings are usually present on early infected leaves. | Chlorosis and vein banding are observed on young quadrifoliates. Terminal bud necrosis is absent. Concentric chlorotic rings are absent. |
| Secondary symptoms | Total necrosis of especially early infected plants at high temperatures. Severe stunting, axillary shoot proliferation with severely distorted leaves. | Total necrosis of infected plants is absent. Axillary shoot proliferation is uncommon. Severe leaf distortion is absent. Leaflets are usually reduced in size showing general chlorosis, vein banding and dark-green patches. |
| Transmission | By thrips in a persistent manner. | By aphids in a persistent manner. |
| Causal virus | Tomato spotted wilt virus | A luteovirus and a symptom-inducing virus, which depends on luteovirus for aphid transmission. |
| Resistant genotypes | No resistance yet detected in cultivated *Arachis* sp. | A group of Virginia runners from northern Ivory Coast are highly resistant. |
| Geographical distribution | Major groundnut-growing countries. | Only in Africa, south of the Sahara. |

[1]Comparison with green rosette is not made because of totally different symptoms of green rosette.



A single lesion isolate from cowpea, which was subsequently maintained in groundnut ('TMV 2') by mechanical sap inoculations produced terminal bud necrosis in summer (March-July) in Hyderabad. However, the same isolate on the same cultivar rarely produced terminal bud necrosis between October and February. When extract from ring mosaic (Narayanasamy *et al.*, 1975) infected groundnut leaflets was mechanically inoculated onto healthy groundnut, it produced symptoms of terminal bud necrosis. In all disease surveys we have noticed several groundnut plants in Coimbatore in September showing typical BND symptoms.

**Causal Virus**

BND was shown to be caused by tomato spotted wilt virus (TSWV) (Ghanekar *et al.*, 1979). The structure of TSWV particles is unique among plant viruses. The virus particles are spherical, surrounded by a lipo-protein membrane. The diameter of the particles ranges from 70 to 90 nm (Fig. 12.1.*6*). In thin sections of infected cells virus particles are scattered between the membranes of the endoplasmic reticulum (Fig. 12.1.*5*).

**Host Range**

TSWV has an extremely wide host range exceeding 200 species in 34 families. On cowpea 'C 152' and 'Early Ramshorn', TSWV produces concentric chlorotic and necrotic lesions on inoculated primary leaves. The virus produces necrotic lesions on *Petunia hybrida* and local lesions followed by systemic infection on *Cucumis sativus*, *Nicotiana tabacum* ('Samsun NN' and 'White Burley'), *N. glutinosa* and *N. clevelandii* × *N. glutinosa* hybrid.

**Transmission**

TSWV can be transmitted by mechanical sap inoculations if extracts are prepared in chilled neutral phosphate buffer containing either mercaptoethanol or $Na_2SO_3$ as antioxidants. The virus is readily graft transmissible and is not transmitted through groundnut seed.

Thrips *Frankliniella schultzei and Scirtothrips dorsalis* transmit the virus in a persistent manner. *F. schultzei* is the most efficient vector and is chiefly responsible for the disease spread in the field. Larvae acquire the virus. Adults cannot acquire the virus but can transmit it (Amin *et al.*, 1981).

**Physical Properties of Virus in Buffered Groundnut Sap**

The infectivity dilution end point is between $10^{-2.5}$ and $10^{-3.0}$, and the thermal inactivation point is between 45 and 50°C. Virus retained infectivity for 4 hr but not for 5 hr at room temperature (30°C).

**Methods for Disease Diagnosis**

1. Positive reaction in enzyme-linked immunosorbent assay (ELISA) employing TSWV antiserum.
2. Presence of typical TSWV particles in infected plant extracts and in thin sections of infected leaves.
3. Chlorotic and necrotic concentric rings on inoculated primary leaves of cowpea and necrotic lesions on *Petunia hybrida*.
4. Terminal bud necrosis and distinct secondary symptoms, especially the presence of concentric rings on leaflets, axillary shoot proliferation and distorted and mottled leaflets on axillary shoots of groundnut.

5. Transmission by thrips.
6. Low thermal inactivation point.

**Management of Disease**

High incidence of BND has been shown to be primarily associated with infestation by immigrant thrips during August-September and January-February (P.W. Amin, unpublished data; Reddy *et al.*, 1983a). Sowings of kharif crop done in the middle of June or with the first onset of monsoons reduce significantly the disease incidence and subsequent crop loss. Crops sown in July show over 50% disease incidence and suffer heavy crop loss in Hyderabad region. Rabi groundnuts show lower disease incidence when sown in November than when sown in December or January (ICRISAT Annual Reports 1978-79, 1979-80, 1980-81; Reddy *et al.*, 1983a).

An increase in plant density decreases the proportion of infected plants. Plant to plant spacing of about 10 cm and row to row of about 40 cm give significantly lower disease incidence than 15 cm × 75 cm plant spacing (ICRISAT Annual Report 1978-80; Reddy *et al.*, 1983a). However, the number of infected plants per unit area remains unaffected.

Carbofuran applied at 1 kg active ingredient (a.i.)/per hectare in the soil at the time of sowing gave a small increase in yield. However, it is not economical. The incidence of BND is reduced by weekly sprays of systemic insecticides such as dimethoate (450 ml a.i./ha). However, the increase in yield did not cover the cost of insecticide application. In addition excessive use of pesticides should not be recommended for environmental reasons (Reddy *et al.*, 1983a).

Narayanasamy and Ramaiah (1976) reported insecticidal control of ring mosaic at Coimbatore. Since ring mosaic is no other than BND the data are included under BND. Field incidence on susceptible checks was lower than 16%. Interestingly disease incidence with the exception of plots treated with chlorfenvinphos was not significantly different from that in susceptible checks. However, the yield increase in various treatments with insecticides was significant. Thus it appears that the significant increase in yield is due to control of pests and not due to reduction in BND incidence.

**Antiviral Chemicals**

Recently Narayanasamy *et al.* (1983) reported that an antiviral principle, which could be precipitated by ammonium sulphate (Narayanasamy and Ramaiah, 1983), was found effective in reducing the BND incidence, and it increased the yields significantly. Although several substances have been reported to inhibit plant virus infectivity, none so far have been used successfully on a field-scale for controlling plant virus diseases. Thus the report by Narayanasamy *et al.* (1983) should be re-examined.

Over 5,000 germplasm lines were screened at the ICRISAT under natural conditions, then the disease incidence in susceptible checks exceeded 60%. None of the germplasm lines were found resistant or immune to the virus. However, North Carolina accessions 343, 841, 1705 1741, 2242 and 'C 123', 'C 145' and 'Robut 33-1' ('Kadiri 3'), although susceptible wher inoculated mechanically in the laboratory, showed significantly lower than average disease incidence (ICRISAT Annual Reports 1980-81, 1981-82) when tested in both kharif and rab seasons. 'Robut 33-1' and 'NCAc 343' are high-yielding cultivars with desirable poc characteristics.

Several germplasm lines reported to be resistant by Ravindranath and Indirá (1975) and



germplasm lines reported to be tolerant by Narayanasamy (1983) were found susceptible when tested under laboratory and field conditions at the ICRISAT.

Among several wild *Arachis* spp. tested, *A. chacoense* (PI 10602) and *A. pusilla* were not infected despite repeated mechanical sap inoculations and by feeding with viruliferous adults of *F. schultzei* (A.M. Ghanekar and P.W. Amin, unpublished data). These 2 species are currently being crossed with high-yielding cultivated groundnut with the hope of evolving BND-resistant groundnut cultivars.

## GROUNDNUT MOSAIC
### (Syn. Bud Necrosis Disease Caused by Tomato Spotted Wilt Virus)

Groundnut mosaic was reported by Nariani and Dhingra (1963). Symptoms were strikingly similar to BND. Since several precautions are required to transmit TSWV by mechanical sap inoculations, the authors failed to transmit groundnut mosaic mechanically. Mosaic was also not transmitted by aphids (*Aphis craccivora* and *A.gossypii*), by whitefly (*Bemisia tabaci*) and by leafhoppers (*Empoasca, Orosius* sp.) Thrips were not tested. Subsequently mosaic was also reported from Tamil Nadu (Kousalya *et al.*, 1970). Symptoms described were similar to those by BND (Kousalya *et al.*, 1974). However, the symptoms described on *Vigna unguiculata* (Linn.) Walp., *Dolichos biflorus* auct. non Linn. *and Phaseolus lathyroides* Linn. were different from those caused by TSWV. Since the authors have not eliminated the possibility of presence of more than one virus in the field-collected samples, limited results of host range cannot be used to regard mosaic as different from BND. Thus the mosaic should be considered as a synonym to BND.

## BUNCHY TOP, CHLOROSIS AND RING MOTTLE
### (Syn. Bud Necrosis Disease Caused by Tomato Spotted Wilt Virus)

Three diseases, bunchy top, chlorosis, and ring mottle were reported by Sharma (1966). Causal agents of none of the 3 diseases were characterized. Symptoms of bunchy top were strikingly similar to secondary symptoms caused by TSWV. The author claimed seed transmission of bunchy top.

Symptoms of chlorosis were identical to the early symptoms of BND on young groundnut quadrifoliates. It is difficult to explain the persistent aphid transmission and seed transmission reported by Sharma (1966).

Ring mottle symptoms described were identical to the chlorotic rings produced by TSWV.

Though all the 3 diseases reported by Sharma (1966) bore striking resemblance to symptoms by TSWV (Costa, 1941, 1950), no comparisons with TSWV were made. Seed transmission in all the 3 diseases is a good indication that the plants raised from seeds of infected plants were infested by viruliferous thrips. It is difficult to prevent thrips contamination in glasshouses. In addition very few seeds were used by Sharma (1966) in his transmission studies. Cross-protection tests reported by the author were inconclusive to consider bunchy top, chlorosis and ring mottle as 3 distinct diseases. Interestingly *Desmodium diffusum* and *Alysicarpus longifolius* Wight & Arn. were infected by the causal agents of all the 3 diseases.

## RING MOSAIC
### (Syn. Bud Necrosis Disease Caused by Tomato Spotted Wilt Virus)

This disease was reported from Coimbatore by Narayanasamy *et al.* (1975). Symptoms were similar to those described by Reddy *et al.* (1968) and Chohan (1972), including terminal bud necrosis. TSWV causing ring mosaic disease and BND were compared at the ICRISAT for host range, and serological reaction with ELISA employing TSWV antiserum produced at the ICRISAT. No serological differences were detected. TSWV causing ring mosaic also produced bud necrosis symptom when tested at the ICRISAT. Ring mosaic was also efficiently transmitted by *Frankliniella schultzei* (P.W. Amin, unpublished data). Thus it would be appropriate to refer ring mosaic as bud necrosis disease.

## PEANUT CLUMP VIRUS DISEASE

Peanut clump disease (PCD) was first reported from India in 1926 (Sundararaman, 1926). Subsequently a similar disease in symptomatology from West Africa was also named as "clump" (Trochain, 1931; Bouhot, 1967). PCD from West Africa was shown to be caused by a virus called peanut clump virus (PCV) (Thouvenel *et al.*, 1974). It is difficult to assess now what disease Sundararaman had reported in 1926. However, the occurrence of a disease similar to West African PCD was reported by Reddy *et al.* (1979) on groundnuts in the Punjab, Gujarat, and Andhra Pradesh. The causal virus was subsequently characterized (Reddy *et al.*, 1983).

### Symptoms

The disease occurs in patches in the field (Fig. 12.2.*1*). In the succeeding years the disease occurs in the same position, usually in progressively enlarged patches. Initially chlorotic ring spots and mottling appear on young quadrifoliates of 2–3-week-old seedlings. Subsequently these symptoms fade away and the leaflets become dark green with faint mottling. Thereafter plants become severely stunted, appear dark green and bushy (Fig. 12.2.*2*). The new quadrifoliates exhibit mosaic mottling and chlorotic rings. Axillary shoot proliferation is not observed. Flowering and peg formation occur on infected plants. However, small pods are produced resulting in seeds of reduced size. Green rosette occurring in West Africa resembles clump in symptoms. However, these diseases differ in all other aspects (Table 12.2).

Infected plants also show marked reduction in the size of the root system. Usually roots become dark-coloured and the outer layer peels off easily which is probably due to the secondary invasion by other organisms.

### Causal Virus

Virus particles are rod-shaped, 24 nm in diameter with 2 predominant particle lengths of 184 nm and 249 nm (Reddy *et al.*, 1983b) when stained with uranyl acetate (Fig. 12.2.*5*). The particle lengths were 165 and 230 nm when stained with neutral phosphotungstate. The molecular weight of the coat protein is 24,000 daltons. The nucleic acid is single stranded with 2 components of molecular weight 1.6 and 2.0 $\times$ $10^6$ daltons.

In thin sections of infected leaves several virus-like particles are observed. They are arranged side by side in layers at with the angle their long axes in adjacent layers alternating to give a 'herring-bone' pattern.




**Fig. 12.2.** *1.* Field on Punjab Agricultural University Farm, Ludhiana, showing clump-infected groundnut plants: *2* Severely stunted groundnut plant infected with peanut clump virus *(right)* and uninfected plant *(left)*, *3 Nicotiana clevelandii* × *N glutinosa* hybrid infected with peanut clump virus isolate from Ludhiana, *4* Veinal necrosis in primary leaves of *Phaseolus vulgaris* ('Topcrop') induced by peanut clump virus isolate from Ludhiana; *5* Purified virus particles of peanut clump (bar represents 90 nm), *6* Clump-infected field on Punjab Agricultural University Farm, Ludhiana; treated with dibromochloropropane *(right)* and untreated block *(left)*

**Table 12.2.** Differences between clump disease and green rosette

| Characteristics | Clump disease | Green rosette |
|---|---|---|
| Symptoms | Dark green clumped plants showing chlorotic rings on young leaflets. | Dark green rosetted plants. Margins of older leaves are rolled outwards. Young leaflets show mild chlorotic mottling and narrow chlorotic streaks. |
| Transmission | Soil-borne. *Polymyxa graminis* transmitted. Easily sap transmissible. | Not soil borne. Aphid transmitted. Only symptom-inducing virus is mechanically transmissible following special precautions. |
| Causal virus | Rod-shaped with two particle lengths. | An aphid-transmitted luteovirus and a symptom-inducing virus which depends on luteovirus for aphid transmission. |
| Geographical distribution | West Africa and India. | Appears to be restricted to West Africa. |

**Host Range**

PCV produces local lesions on *Chenopodium quinoa, Cyamopsis tetragonoloba* (Linn.) Taub. and *Vigna unguiculata* and systemic reaction on *Nicotiana benthamiana, N. clevelandii,* and *N. clevelandii* × *N. glutinosa* hybrid (Fig. 12.2.*3*). A characteristic systemic veinal necrosis produced on *Phaseolus vulgaris* (Frenchbean cv. local) (Fig. 12.2.*4*) and local lesions on *Canavalia ensiformis* (Linn.) DC. could be used for the diagnosis of the different isolates of the PCV in India.

**Transmission**

The virus is readily transmissible by sap and by grafting. Healthy groundnut seeds sown in soil from clump-infested fields collected at depths between 10 and 25 cm produced seedlings with typical disease symptoms. Soil samples dried at 37°C for 1 week also produced seedlings with PCD. The phycomycete *Polymyxa graminis* has been found to be associated with the disease. Graminaceous hosts which support *P. graminis* and PCV multiplication have been identified. Roots from these hosts containing *P. graminis* when incorporated into sterile soil reproduced the disease on groundnut. Evidence accumulated so far indicates that PCV is transmitted by *P. graminis* (B.L. Nolt, unpublished data).

**Physical Properties of Virus in Buffered Groundnut Sap**

The thermal inactivation point is between 60 and 65°C and the dilution end point between $10^{-3}$ and $10^{-4}$. The virus remained infective for over 20 days at room temperature (25-30°C).

**Methods for Disease Diagnosis**

1. Disease occurs year after year in the same patch.
2. Presence of typical PCV particles in infected plant tissue.
3. Typical symptoms on groundnut.
4. Chlorotic local lesions on inoculated primary leaves of cowpea, necrotic local lesions on sword beans and veinal necrosis on Frenchbeans.



**Management of Disease**

*Application of soil biocides.* Several soil biocides such as nemagon (1, 2-dibromide, 3-chloropropene), carbofuran, and temik effectively control the disease (Fig. 12.2.*6*). However, these biocides are not economical.

*Host-plant resistance.* The most desirable way to control a soil-borne virus disease is by growing resistant cultivars. Over 4,000 germplasm lines have been screened on the Punjab Agricultural University Farm in Ludhiana (Fig. 12.3.*1,2*) and 2,500 germplasm lines on the Andhra Pradesh Agricultural University Farm in Bapatla. None of the germplasm lines were found either immune or resistant to PCV.

Since the virus is seed-borne, seed from clump-infested areas should not be used for planting.

Groundnut crop sown during the rabi season in November and December escapes the disease.

## PEANUT MOTTLE VIRUS DISEASE

Peanut mottle was first reported from the USA (Kuhn, 1965). Subsequently the virus has been recorded in all the groundnut-growing countries including India (Reddy *et al.*, 1978).

**Symptoms**

Initially dark-green islands interspersed with chlorotic areas appear on the youngest quadrifoliates (Fig. 12.4.*1*). Symptoms are clearly apparent when viewed against transmitted light. Interveinal depression and upward rolling of edges are also observed in certain genotypes. Infected plants are not markedly stunted although the size of the leaflets is reduced compared to uninfected plants. Older plants rarely show typical disease symptoms. Peanut mottle can cause yield reductions up to 60%.

**Causal Virus**

Peanut mottle virus (PMV) belongs to the potato virus Y group. Virus particles are flexuous rods about 750 nm in length and 12 nm in width (Fig. 12.4.*3*). The virus produces pinwheel and cylindrical type of inclusions in the cytoplasm of infected cells (Fig. 12.4.*4*).

**Host Range**

Host range is mostly restricted to legumes. PMV produces dark-brown necrotic local lesions on *Phaseolus vulgaris* 'Topcrop' (Fig. 12.4.*2*) and systemic mosaic symptoms on cowpea, pea and soybean.

**Transmission**

PMV is transmitted by *Aphis craccivora, A. gossypii, Myzus persicae, Rhopalosiphum padi* and *R. maidis* in a non-persistent manner. It is readily transmitted by mechanical sap inoculations and grafting. The virus is also seed transmitted from 0 to 4% depending on the cultivar.

**Physical Properties of Virus in Buffered Groundnut Sap**

The thermal inactivation point is between 55 and 60°C and the dilution end point between $10^{-2}$ and $10^{-3}$. The virus retained infectivity at 20°C for 2 days but not for 3 days.

**Fig. 12.3.** *1.* A section of trial on screening for resistance to peanut clump disease on Punjab Agricultural University Farm, Ludhiana; *2.* Germplasm accession 'NC Ac 17866', tolerant to peanut clump disease.



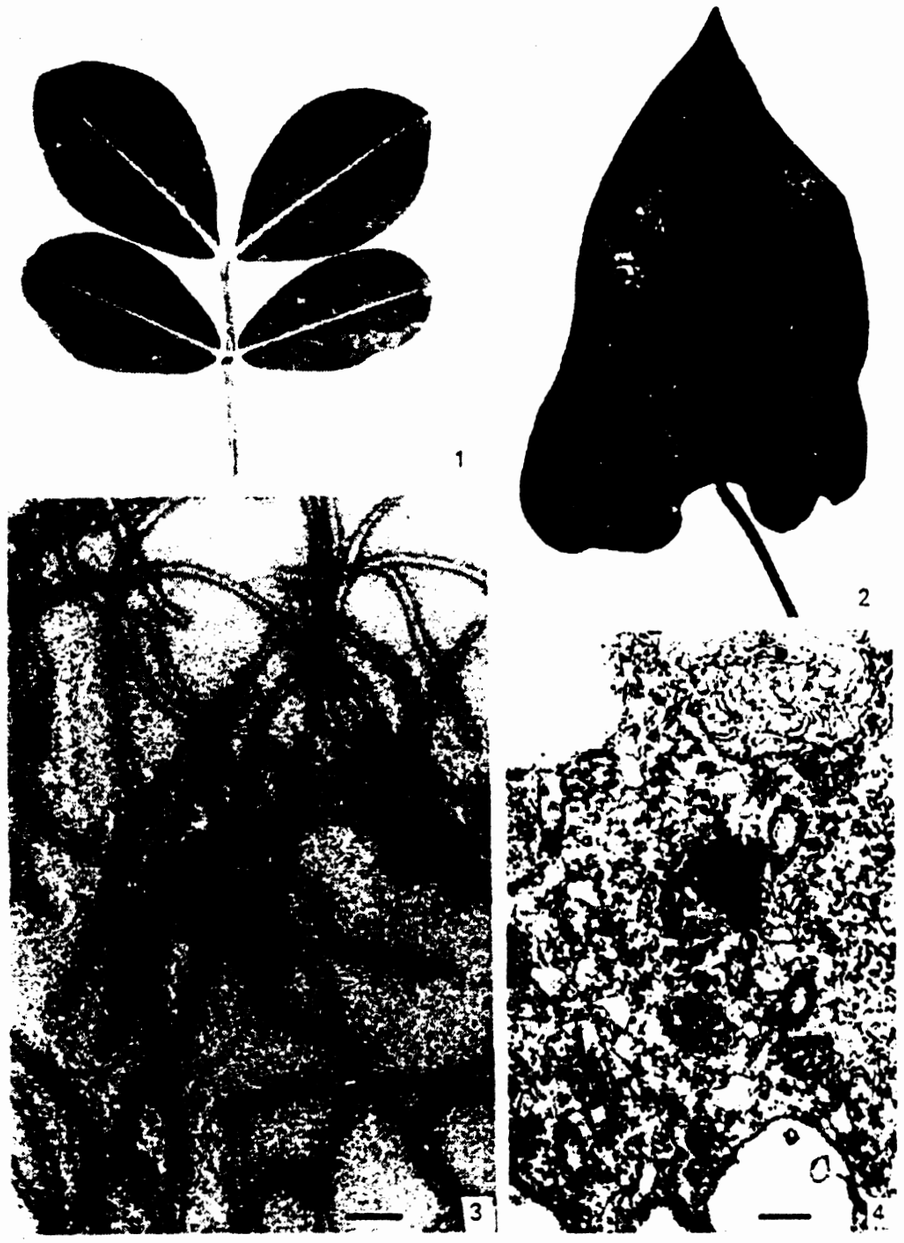

**Fig. 12.4.** *1.* Young leaf with symptoms of peanut mottle disease; *2.* Primary leaf of *Phaseolus vulgaris* ('Topcrop') with necrotic lesions induced by peanut mottle virus; *3.* Purified peanut mottle virus (bar represents 72 nm); *4.* Electron micrograph of a thin section of leaflet infected with peanut mottle virus showing cylindrical inclusions (bar represents 265 nm).

**Methods for Disease Diagnosis**

1. Positive serological reaction with PMV antisera in microprecipitin, precipitin ring and ELISA tests (Rajeshwari *et al.*, 1983).
2. Necrotic local lesions on *Phaseolus vulgaris* ('Topcrop') beans.
3. Typical symptoms on groundnut which include mosaic mottling, interveinal depression and upward rolling of leaf edges.
4. Presence of flexuous rod-shaped virus particles in infected plants.
5. Non-persistent transmission, i.e., the virus is acquired in short acquisition access periods of 30-150 sec and can be transmitted immediately by aphids.

**nagement of Disease**

1. Seeds from infected plants should not be used for planting. The primary source of inoculum appears to come from seed transmitted plants. Thus cultivars with no-seed transmission are likely to reduce the primary source of inoculum. Tests conducted at the ICRISAT indicate that over 20,000 kernels of each of the genotypes 'EC 76446 (292)', and 'NCAc 17133 RF' have not shown any seed transmission. Currently efforts are being made to transfer no-seed transmission characteristic into locally grown cultivars.
2. It is essential to grow a groundnut crop at a distance of 100 m or more from other highly susceptible legumes such as soybeans, cowpeas and navybeans, especially from the latter 2 hosts in which PMV can be seed transmitted (Demski and Kuhn, 1983).
3. Several germplasm lines have been screened for resistance to PMV at the ICRISAT. None of them were found to be resistant. However, 'NC Ac 2243' showed no reduction in yield following infection. Currently efforts are being made at the ICRISAT to produce cultivars tolerant to PMV coupled with non-seed transmission.
4. Two wild *Arachis* spp., *Arachis chacoense* and *A. pusilla*, have been found resistant to PMV and cytogeneticists at the ICRISAT are making efforts to transfer this resistance to the cultivated groundnut.

## GROUNDNUT ROSETTE
### (Syn. Bud Necrosis Disease Caused by Tomato Spotted Wilt Virus)

It was first reported from India by Bisht *et al.* (1963). Its symptoms described by them resembled BND symptoms with the exception of the terminal bud necrosis. It is difficult to explain the aphid transmission data by the authors. It was claimed to be of 'Persistent type', but there are no data to support this claim. It is likely that the transmission reported may have been due to external infestation by thrips. Singh and Gupta (1968) reported a disease called 'rosette' from Rajasthan. Symptoms on groundnut were not described. However, Mathur *et al.* (1971) described the symptoms of a 'rosette' disease on groundnut from Rajasthan which resembled BND.



Kousalya *et al.* (1967) reported 'rosette' from Tamil Nadu and it was found to be transmitted by *Aphis craccivora* (Kousalya *et al.*, 1970). Thrips were not tested in the transmission experiments. It is difficult to interpret their results because the virus reported has not been subsequently characterized. In disease surveys in Tamil Nadu, which included Coimbatore district from where 'rosette' was earlier reported, we have so far been able to observe only bud necrosis disease. Symptoms of 'rosette' described by Kousalya *et al.* (1967 and 1973) resembled BND. It is also difficult to interpret the positive correlation of aphid population to rosette disease (Kousalya *et al.*, 1971 a,b). We have so far not been able to collect groundnuts showing BND symptoms where the causal virus could be transmitted by *A.craccivora*.

## COWPEA MILD MOTTLE VIRUS

The natural occurrence of cowpea mild mottle virus (CMMV) has been reported from India (Iizuka *et al.*, 1984). Its incidence is low and currently is regarded as a minor virus disease.

**Symptoms**

Newly formed leaves, about 2 weeks after inoculation, show vein clearing followed by downward rolling (Fig. 12.5.*1*). Subsequently necrosis of leaves and petioles occurs which leads to dropping of leaves. Plants are severely stunted and rarely produce any pods. Under field conditions infected plants are severely stunted with older leaves showing necrosis and younger vein-banding symptoms. The disease is usually present on rabi groundnuts.

**Causal Virus**

The virus particles are slightly flexuous rods of 15 nm diameter (Fig. 12.5.*3*) with a model length of 610 nm. The molecular weight of the coat protein is 33,000 daltons. The nucleic acid is ribose type, single stranded, with a molecular weight of $2.6 \times 10^6$ daltons. CMMV is found to be serologically related to several carlaviruses (Rajeshwari *et al.*, personal communication).

**Host Range**

The virus produces local lesions on *Beta vulgaris, Cajanus cajan, Chenopodium amaranticolor, C. quinoa* and *Cyamopsis tetragonoloba* and systemic symptoms on *Canavalia ensiformis, Cassia occidentalis, Glycine max* (Fig. 12.5.*2*), *Nicotiana clevelandii, Phaseolus vulgaris, Pisum sativum* and *Vigna unguiculata*.

**Transmission**

CMMV is easily sap transmissible. It is transmitted by the whitefly *Bemisia tabaci* in a non-persistent manner. It is seed transmitted in soybeans. There is no evidence for seed transmission in groundnut.

**Physical Properties of Virus in Buffered Soybean Extract**

The thermal inactivation point is between 75° and 80°C. The virus remained infective at room temperature (25°-35°C) for over 8 days.

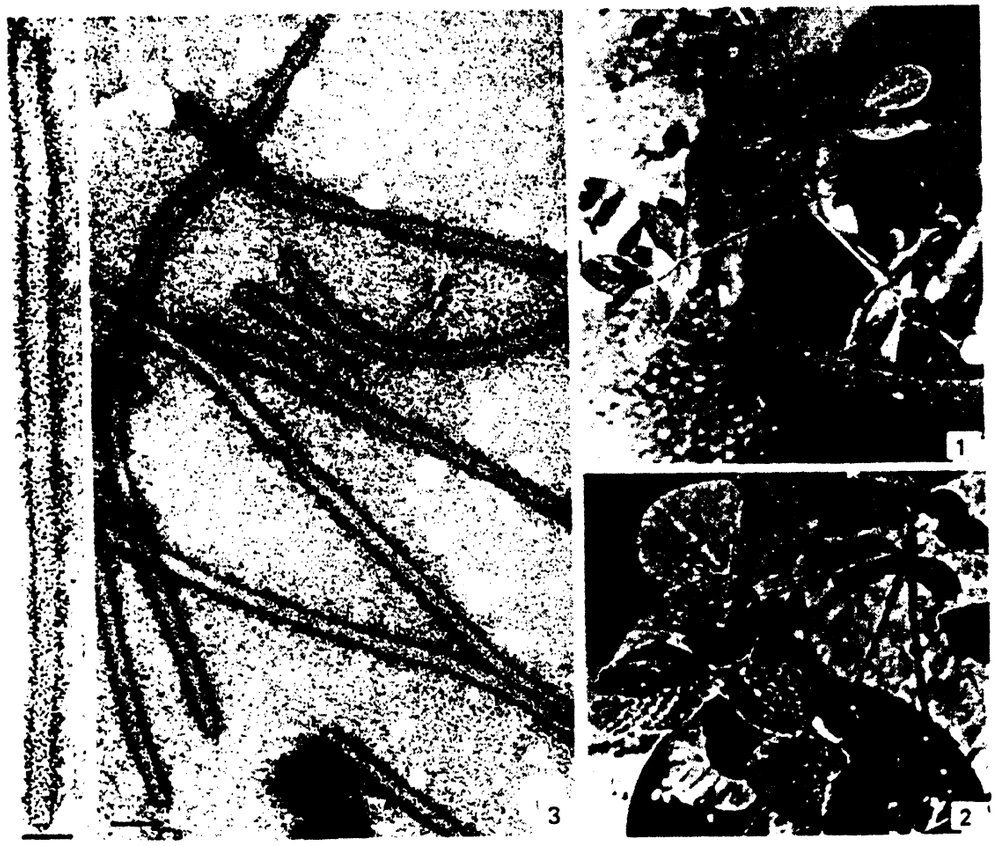


**Fig. 12.5.** *1.* Vein banding and leaf rolling induced by cowpea mild mottle virus in groundnut; *2.* Vein clearing and mosaic induced by cowpea mild mottle virus in soybean ('Bragg'); *3.* Electron micrograph of purified cowpea mild mottle virus (bar represents 46 nm) (inset bar represents 26 nm).

**Methods for Disease Diagnosis**

1. In ELISA and in immunosorbent electron microscopy tests virus present in crude extracts and purified preparations reacts positively with CMMV antiserum (Rajeshwari *et al.*, personal communication).
2. Non-persistent transmission by *Bemisia tabaci*. The virus could be acquired in less than 10 minutes and transmitted soon after acquisition without any detectable latent period (Muniyappa and Reddy, 1983).
3. Presence of slightly flexuous rod-shaped virus particles.
4. The virus produces chlorotic local lesions on *Chenopodium quinoa* and necrotic lesions on *Beta vulgaris*.
5. Typical symptoms on groundnut leaflets which include vein banding, downward leaf rolling and necrosis.
6. High thermal inactivation point.



**Management of Disease**

The disease is of minor importance on groundnut though it appears to be economically important on other legumes, soybeans, cowpeas, etc. Therefore, no control measures are recommended.

## PEANUT GREEN MOSAIC

Peanut green mosaic caused by peanut green mosaic virus (PGMV) has been reported from Tirupati, Andhra Pradesh (Sreenivasulu *et al.*, 1981). PGMV produces chlorotic spots and vein clearing on young quadrifoliates followed by severe mosaic. Plants are severely stunted.

PGMV belongs to potato virus Y group. Virus particles are flatuous rods of about 750 nm in length. Cylindrical inclusions are present in the cytoplasm of plants. PGMV is not serologically related to peanut mottle virus.

The virus is mechanically transmissible to 16 species of Leguminosae, Solanaceae, Chenopodiaceae, Aizoaceae and Pedaliaceae.

PGMV is readily sap transmissible. It is also transmitted by *Aphis gossypii* and *Myzus persicae* in a non-persistent manner. The virus is not seed transmitted.

PGMV remained infective in buffered groundnut sap for 3 to 4 days at 25°C. The thermal inactivation point is between 55°C and 60°C.

PGMV in crude plant extracts and purified preparations reacts with PGMV antiserum in Ouchterlony gel double diffusion, hemagglutination and in ELISA tests. The virus also could be diagnosed on the basis of necrotic local lesions on the primary leaves of *Phaseolus vulgaris* (Frenchbean cv. local) and on *Cassia obtusifolia*.

Since PGMV appears to be restricted in its distribution no control measures have been investigated.

## GROUNDNUT CHLOROTIC SPOT VIRUS

Groundnut chlorotic spot virus (GCSV) was reported from Tirupati by Haragopal and Nayudu (1971) but the virus was not characterized. Interestingly the physical properties reported by the authors were similar to TSWV. However, the symptoms described differed from BND. Later GCSV material brought to the ICRISAT for characterization was renamed as peanut green mosaic because of the conspicuous mosaic symptoms. In addition chlorotic spots are induced by several viruses occurring in groundnut. However, the host range and physical properties reported by Haragopal and Nayudu (1971) for GCSV differed from PGMV. Since Haragopal and Nayudu (1971) used field material, and not an isolate extracted from a single lesion, GCSV was probably a contaminant occurring with PGMV. Interestingly symptoms of GCSV on *Arachis hypogaea, Pisum sativum* and *Glycine max* resembled peanut mottle virus.

## YELLOW MOSAIC DISEASE

A yellow mosaic disease transmitted by the whitefly *Bemisia tabaci* was reported from Andhra Pradesh (Sudhakar Rao *et al.*, 1980). Infected leaflets showed bright-yellow patches. They were puckered and the edges were curled upwards.

The disease was transmitted by *B. tabaci* and grafting. The disease was also transmitted by *B. tabaci* from groundnut to several other legumes like *Phaseolus aureus* Roxb., *P. mungo*, *Cajanus cajan* (Linn.) Millsp., etc.

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