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Abstract

The first known instance of tobacco streak virus (TSV) in groundnut (*Arachis hypogaea* L.) was recorded when it caused a disease epidemic in the year 2000 in Anantapur, Andhra Pradesh, India. The disease was named as peanut stem necrosis disease (PSND). It is difficult to distinguish between PSND and peanut bud necrosis disease (PBND), another economically important virus disease of groundnut based on symptoms alone. Techniques have been developed for precise diagnosis of the disease by ELISA and by the reaction of indicator hosts. TSV infects several economically important crop plants and survives on many weed hosts under field conditions. Parthenium, a widely distributed and symptomless carrier of TSV, plays a major role in the perpetuation and spread of the disease. While the role of infective pollen and flower-inhabiting thrips in the transmission of TSV has been established, seed transmission of TSV in groundnut and other crop plants as well as in weed hosts requires further investigation. However, based on field observations and laboratory tests, this bulletin suggests several interim measures for the management of the disease.

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Peanut Stem Necrosis: A New Disease of Groundnut in India

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Peanut Stem Necrosis: A New Disease of Groundnut in India

Introduction

A disease epidemic resulting in the death of young groundnut (Arachis hypogaea L.) plants occurred in the rainy season of the year 2000 in Anantapur district, Andhra Pradesh, where the crop is usually grown on 0.7 million hectares (ha). The disease affected nearly 225,000 ha and the crop losses were estimated to exceed Rs 3 billion (US\$65 million). Initially, the disease was suspected to be peanut bud necrosis disease (PBND) caused by peanut bud necrosis tospovirus (PBNV) because of the characteristic necrosis of terminal buds in the plants. In subsequent studies however, tobacco streak ilarvirus (TSV) was found associated with the disease (Reddy et al. 2002). The disease was named as 'Peanut Stem Necrosis Disease' (PSND). This was the first report of occurrence of TSV in groundnut in India. TSV was also shown to cause sunflower necrosis disease in sunflower (Prasada Rao et al. 2000, Ravi et al. 2001, Ramiah et al. 2001, Bhat et al. 2002 b). Although PSND and PBND are caused by two distinct viruses belonging to the Ilarvirus and Tospovirus groups respectively, the symptoms produced by them on groundnut are very similar. It is often difficult to distinguish between these two diseases in the later stages based on symptoms alone.

Distribution

PSND occurs in the districts of Anantapur, Kurnool, Cuddapah and Chittoor in Andhra Pradesh and Raichur in Karnataka. Limited surveys carried out in Gujarat and Maharashtra did not show the presence of the disease in surveyed areas (Porbandar, Rajkot, Junagadh in Gujarat and Jalgaon and Dhulia in Maharashtra). However, more extensive surveys are needed in Karnataka, Tamil Nadu and Maharashtra, where sunflower necrosis disease caused by TSV is prevalent in sunflower crop.

Symptoms

Symptoms in groundnut first appear on young leaves as necrotic lesions and veinal necrosis (Fig. 1). The necrosis later spreads to the petiole and stem. Necrotic lesions on the stem later spread upwards killing the bud (Fig. 2). Majority of the plants infected within a month after sowing die due to necrosis, which also spreads downwards in case of early infection. In some cultivars, plants that survive infection show proliferation of axillary shoots. The leaflets on these axillary shoots are small and show

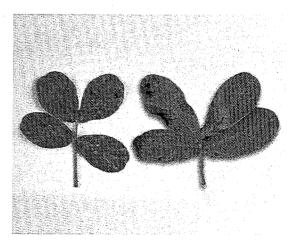


Figure 1. Groundnut leaflets showing necrotic lesions and veinal necrosis due to TSV infection.

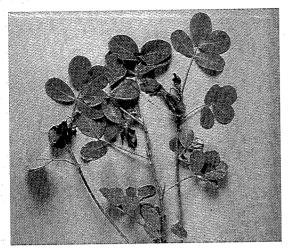


Figure 2. Groundnut plant infected with TSV showing necrosis of the terminal leaflets.

general chlorosis unlike the secondary symptoms of PBNV, where distortion and mosaic mottling of leaf lamina are common. In some cultivars, pods harvested from the PSND infected plants show necrotic lesions (Fig. 3).

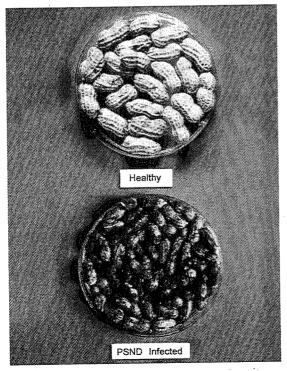


Figure 3. Pods from healthy (top) and PSND diseased groundnut plants (below).

Causal Virus

The virus particles are spherical, in the range of 25-35 nm in diameter (Fig. 4). The virion has a 28 kDa protein capsid subunit and a tripartite genome of 3.7, 3.1, 2.2 kb with a 0.9 kb subgenomic RNA.

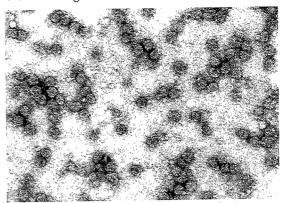


Figure 4: Virus particles of tobacco streak virus.

Host Range

The virus can infect many cultivated plant and weed species under experimental and natural conditions.

Experimental host range

Plant hosts infected by TSV on mechanical inoculation are presented in Table 1. The virus infected Amaranthus viridis, Commelina benghalensis, Parthenium Trianthema and hysterophorus portulacastrum without producing any symptoms. The following species, Brassica oleracea var. capitata and botrytis, Capsicum annuum, Cardiospermum helicacabum, Cucumis sativus, Datura muricata, Hyptis brevipes, Lagenaria cineraria, Lycopersicon esculentum, Pisum sativum, Physalis minima, Raphanus sativus, Sida acuta, Spinacia oleracea, and Zea mays did not get infected on mechanical inoculation.

Table 1. Reaction of various plant hosts to mechanical inoculation of TSV

	Symptoms ^a		
Host species	Primary	Systemic	
Abelmoschus esculentus	NL	LN, D	
Ageratum conyzoides	NL	LN	
Amaranthus viridis	None	SL	
Cajanus cajan	NL	TN of leaves and stem	
Cicer arietinum	NL		
Commelina benghalensis	None SL		
Crotalaria juncea	CL TN of leaves and stem		
Chenopodium quinoa	CL		
C. amarnticolor	CL	TN of leaves and stem	
C. murale	CL	CL	
Cyamopsis tetragonoloba	NL	NL .	
Datura stramonium	SL	MO	
Dahlia rosea	CL/ NL	LN, D	
Glycine max	CL/ NL	TN of leaves and stem	
Gossypium hirsutum	CL	LC	
Gomphrena globosa	CL	VN	
Helianthus annuus	CL	TN of leaves and stem	
Lagasca mollis	None	LN	
Luffa acutangula	CL/ NL		
Momordica charantia	CL/ NL CL/ NL	None	
Nicotiana benthamiana		None	
Nicotiana oeninamiana N. glutinosa	CL MO VC VC		
N. rustica		VC	
	CL	VN	
N. tabacum cv. White Burley	CL	MO	
N. tabacum cv. Havana	CL	TN of leaves	
N. tabacum cv. Turkish	CL	CL	
N. tabacum cv. Samsun	CL	VN	
Parthenium hysterophorus	None	SL	
Phaseolus vulgaris cv. Top Crop	NL, VN	TN of leaves and stem	
Physalis floridana	CL	TN of leaves and stem	
Tagetes erecta	NL	NL	
Trianthema portulacastrum	None	SL .	
Trigonella foenum-graecum	NL	VN of leaves	
Vicia faba	NL	NL	
Vigna unguiculata	NL, VN	TN of leaves and stem	
Vigna mungo cv. ML 267	NL	TN of leaves and stem	
Vigna radiata cv. LBG 20	NL	TN of leaves and stem	

a = Source for mechanical inoculations was groundnut; virus infection confirmed in ELISA.

CL = Chlorotic lesions, D = Death, LC = Leaf curling, LN = Leaf necrosis, MO = Mosaic symptoms, NL = Necrotic lesions, SL = Symptomless, TN = Total necrosis, VC = Veinal chlorosis, VN = Veinal necrosis.

Natural host range

In the ELISA conducted on several weed species collected from groundnut fields around Anantapur, Acalypha indica. Abutilon indicum, Achyranthes aspera, Calotropis gigantea, Cleome viscosa, benghalensis. Croton Commenlina sparsiflorus, Digera arvensis, Euphorbia hirta, Euphorbia geniculata, Lagasca mollis, Leucas aspera, Parthenium hysterophorus and Tridax procumbens tested positive for TSV. Parthenium is a widely distributed weed along the roadsides, in fallow lands and on field bunds. Weed species, Ammannia baccifera, Argemone mexicana, Aristolochia braocteata, Boerhaavia diffusa, Corchorus olitorius, Lantana sp., Ocimum basilicum, Physalis minima, Portulaca sp., and Tephrosia procumbens tested negative for TSV. Among the crop plants, the virus was detected in groundnut, safflower (Carthamus tinctorius), sunflower (Fig. 5),



Figure 5. Symptoms of tobacco streak virus on sunflower.

cotton (Gossypium hirsutum), cowpea (Vigna unguiculata), urdbean (V. mungo), mungbean (V. radiata) and marigold (Tagetes erecta) (Fig. 6). No TSV was detected in chilli (Capsicum annuum), cucumber (Cucumis sativus), ridged gourd (Luffa acutangula), tomato (Lycopersicon esculentum) and brinjal (Solanum melongena).

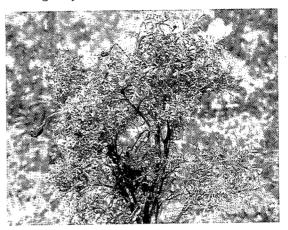


Figure 6. Marigold showing symptoms of tobacco streak virus.

Transmission

In experimental transmission tests with thrips, typical veinal necrosis symptoms were observed on cowpea plants three days after the release of Frankliniella schultzei. Scirtothrips dorsalis and Megalurothrips usitatus onto leaves dusted with pollen from marigold and sunflower, infected parthenium plants. F. schultzei was more efficient than the other two species in disease transmission. The thrips fed on infected leaves alone did not transmit the virus. The disease transmission under natural conditions seems to occur through wounding of leaf tissue as well as infected pollen and their proximity during thrips feeding, rather than a specific virus-vector interaction (Sdoodee and Teakle 1987).

Virus Purification

The inoculated primary leaves of cowpea cv. C 152 or french bean cv. Top Crop showing necrotic lesions and veinal necrosis are most suitable for purification.

Purification procedure

- 1. Collect 100 g inoculated primary leaves showing necrotic lesions.
- 2. Blend the leaves in 400 ml 0:1 M potassium phosphate (KPO₄) buffer (pH 8.0), containing 0.75% monothioglycerol (V/V) and 0.17% diethyl dithiocarbamate (W/V). Use 4 ml buffer for each g of tissue.
- 3. Filter through two layers of cheese cloth.
- 4. To the filtrate, add cold chloroform to give 10% (V/V) and emulsify by thorough mixing for 5 to 10 min.
- 5. Centrifuge at $6000 \times g$ for 10 min at 5°C.
- 6. Collect the aqueous phase (upper clear layer).
- 7. Add sodium chloride (NaCl) to the aqueous phase to give 0.2 M, and to this solution, add polyethylene glycol (PEG, molecular weight 6000-8000) to give 8% (V/W). Stir at 5°C until NaCl and PEG are dissolved, then leave at 5°C for 90 min.
- 8. Collect the pellet by centrifuging at $11,000 \times g$ for 10 min.
- Resuspend the pellet in 150 ml of 0.05 M KPO₄ buffer (pH 8.0) containing 0.2% Triton X-100.
- 10. Clarify at $6000 \times g$ for 10 min at 5°C.
- 11. Layer 26 ml of virus suspension on 12 ml of 30% sucrose cushion solution in SW 28 rotor tubes. Centrifuge for 90 min. at $95,000 \times g$.
- 12. Discard supernatant. Dissolve pellets in 20 ml of 0.05 M KPO₄ buffer (pH 8.0).

- Centrifuge for 10 min at $10,000 \times g$.
- 13. Prepare sucrose gradients in a Beckman SW 28 rotor tube by layering 6, 9, 9, 9 ml of 10, 20, 30 and 40% sucrose, respectively, in 0.01 M KPO₄ buffer. Allow sucrose solution to form a gradient by leaving them overnight in a refrigerator at 4°C.
- 14. Layer 5 ml of supernatant from step 12 on each sucrose gradient and centrifuge at 4° C at $90,000 \times g$ for 1.5 hours (h).
- 15. Draw light scattering zones at a depth of 50 to 56 mm and 58 to 64 mm from the bottom of the tube.
- 16. Dilute the zones in 0.01 M KPO₄ buffer and centrifuge in a Beckman R50 rotor at $96,000 \times g$ for 1.5 h to pellet the virus.
- 17. Resuspend the pellets in small quantity (150-200 ml) of 0.01 M KPO₄ buffer for further use.

Antiserum Production

- 1. Suspend 100 to 150 mg of purified virus in 0.3 ml of 10 mM phosphate buffer (pH 8.0) with an equal volume of Freund's complete adjuvant.
- 2. Obtain a thick emulsion by repeatedly drawing into a syringe and ejecting with force.
- 3. Inject this emulsion sub-cutaneously at multiple sites to a New Zealand white inbred rabbit.
- 4. Subsequently give 4 intra-muscular and sub-cutaneous injections alternatively at multiple sites at weekly intervals using Freund's incomplete adjuvant.
- 5. Bleed the rabbit 2 weeks after the last injection and subsequently at weekly intervals.
- 6. Test the titer of the antiserum using ELISA.

A rabbit can be bled 6-8 times and each bleeding usually yields 10-15 ml of blood serum. Lyophilize the antiserum in small portions (1 ml) and store at -70°C.

Disease Diagnosis

The symptoms of TSV infection are often confused with those caused by PBNV. Therefore, identification of infection by TSV should not be based solely on observation of necrotic symptoms. The following methods can be used for the detection of TSV.

Enzyme linked immunosorbent assay (ELISA)

Several ELISA procedures for virus detection are available. The simplest is the direct antigen coating (DAC) procedure (Hobbs et al. 1987). The detailed step-wise DAC-ELISA procedure for the detection of TSV is given below.

Solutions required

- 1. 0.05 M sodium carbonate buffer, pH 9.6 (Coating buffer): Add 1.59 g Na₂CO₃ and 2.93 g NaHCO₃ to 800 ml of distilled water and make up the volume to 1 liter (L). The pH of the buffer will be 9.60 and there is no need to adjust it. Then add sodium diethyl dithiocarbamate at 0.01 M concentration (1.71 g for 1 L).
- Phosphate buffer saline Tween (PBS-Tween), pH 7.4: Dissolve 8.0 g NaCl, 0.2 g KH₂PO₄, 2.9 g Na₂HPO₄, and 0.2 g KCl in 800 ml of distilled water and make up the volume to 1 L.
- 3. Conjugate buffer or Antibody buffer: To 100 ml of PBS-Tween add 2 g of polyvinyl pyrrolidone (PVP) to give a 2%

- concentration, and 0.2 g of albumin togive 0.2% concentration.
- 4. Substrate buffer: Dissolve 97 ml of diethamolamine in 800 ml of water. Add concentrated HCI to adjust the pH to 9.8 and then make up the volume to 1 L. This solution can be stored but pH should be adjusted to 9.8 prior to each use.

Procedure

- i. Prepare plant extract in coating buffer. It is preferable to use dilution of 1:50. Use appropriate controls such as a buffer, healthy leaf tissue, leaf tissue from TSV-infected plant (positive control) and leaf tissue from any other virus-infected plant (negative control). Incubate the plate at 37°C for 1 h. Dispense 200 µl of plant extract into each well of ELISA plate using a micropipette.
- ii. Pour out the plant extract and rinse the ELISA plate in PBS-Tween. Follow this by washing the plate in three changes of PBS-Tween, allowing 3 min for each wash.
- iii. It is always preferable to use antiserum (TSV) cross-absorbed with healthy plant extract. Grind healthy leaves in conjugate buffer to give a 1:20 dilution. Filter the extract through two layers of cheese cloth. Using this extract, prepare an appropriate dilution of antiserum (high titer antisera can be used at 1:10,000 dilution and low titer antisera are used at 1:500 to 1:2000 dilutions). Incubate the extract containing antiserum for 45 min at 37°C.
- iv. Add 200 µl of the above cross-absorbed antiserum to each well and incubate the ELISA plate at 37°C for 1 h.
- v. Wash the plate in PBS-Tween as in step 'ii'.

vi. Dilute Alkaline Phosphatase-labeled anti-rabbit Ig G or Fc to 1:5,000 or 1:10,000 in antibody buffer. Dispense 200 µl into each well and incubate the ELISA plate at 37°C for 1 h.

vii. Wash the plate in PBS-Tween as in step 'ii'. viii. Dissolve 15 mg tablet of p-nitrophenyl phosphate (5, 15 or 40 mg tablets available) in 30 ml of diethanolamine solution (0.5 mg ml⁻¹). Dispense 200 μl of this substrate mixture into each well and incubate at room temperature for 30 min to 1 h. Record the yellow color development visually. Further development of yellow color due to the production of p-nitrophenol can be stopped by the addition of 50 μl of 3 M NaOH well⁻¹. Take absorbance of yellow color of p-nitrophenol at 405 nm in an ELISA reader.

Indicator host reactions

ELISA is a rapid method and gives precise results. However, if the necessary expertise and facilities are not available for ELISA, reaction on diagnostic hosts such as cowpea cv. C 152 and french bean cv. Top Crop can help to differentiate TSV from PBNV. Both of these viruses produce characteristic local lesions. The procedure for inoculation of diagnostic host is described below.

- Use young leaflets showing characteristic virus symptoms to prepare extract in 0.05 M phosphate buffer (pH 7.0) containing 0.01 M sodium sulfite (1.26 g L⁻¹).
- ii. Triturate tissue in chilled buffer at 1:9 dilution ie, 1 g tissue in 9 ml buffer. Filter the extract through two layers of cheese cloth.
- iii. Select cowpea cv. C 152 or french bean cv. Top Crop plants with fully expanded

- primary leaves.
- iv. Keep the plants in a dark place for 2-3 hours prior to inoculation.
- v. Dust sparingly the primary leaves of cowpea or french bean with an abrasive such as carborundum 600 mesh.
- vi. Hold the leaves with your left palm and apply the plant extract using the folded cheese cloth.
- vii.Rinse the leaves with distilled water immediately after inoculation.

On inoculated primary leaves of cowpea and french bean, TSV produces necrotic local lesions and veinal necrosis within three days whereas PBNV produces only concentric chlorotic/necrotic local lesions five days after inoculation (Fig. 7, 8).

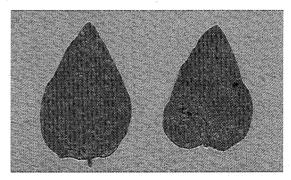


Figure 7. Primary leaves of cowpea (Vigna unguiculata) cv. C 152 inoculated with PBNV (left) and TSV (right).

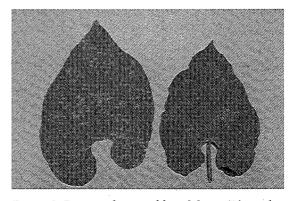


Figure 8. Primary leaves of french bean (Phaseolus vulgaris) cv. Top Crop inoculated with PBNV (left) and TSV (right).

Epidemiology

The PSND occurred at a high incidence in a short period of two weeks covering nearly 225,000 ha of groundnut crop grown during the rainy season in the year 2000 in Anantapur district. Its incidence in the crop was relatively high near field bunds and wastelands. This observation suggested that the source of disease was from outside, such as the weeds growing on bunds or wastelands. The most commonly occurring weeds were Parthenium hysterophorus, Abutilon indicum, Ageratum conyzoides, sparsiflorous, Commelina benghalensis. Cleome viscosa, Euphorbia hirta. Lagasca mollis and Tridax procumbense, all of which were infected by TSV. Parthenium was widely distributed and occurred at all the locations where PSND was recorded (Fig. 9). No symptoms were noticed in TSV-infected parthenium. Although the three thrips species (F. schultzei, M. usitatus and S. dorsalis) were experimentally shown to transmit TSV in the presence of infective pollen, the flowerinhabiting F. schultzei played a major role in the field spread of the virus. F.schultzei collected from flowers of infected parthenium plants carried 8-10 pollen grains on their bodies. When these thrips attacked



Figure 9. Parthenium in full bloom growing in a fallow land.

groundnut plants, the pollen grains got dislodged from their bodies and deposited on the leaves. Thrips, during their feeding, cause injury to both leaf tissue and deposited pollen and thus facilitate virus infection of the plant.

Parthenium produces several flushes during its life cycle, thus ensuring continuous supply of pollen. Heavy westerly winds that occur during August and September can facilitate deposition of pollen on groundnut plants from infected parthenium as well as crop plants such as sunflower and marigold. These pollen can facilitate virus transmission when plants are colonized by the thrips (Prasada Rao et al. 2003). In laboratory tests, transmission was achieved with pollen from sunflower, marigold and parthenium deposited on the groundnut leaves that were colonized by the thrips.

Early infected groundnut plants do not flower. As it is a self-pollinated crop, groundnut is unlikely to contribute to disease spread. Sunflower is often grown adjacent to groundnut crop in peninsular India. Early infected sunflower plants usually produce malformed heads with few or no pollen. However, late-infected sunflower plants produce flowers that could serve as a source of inoculum. Groundnut crop grown adjacent to TSV-infected sunflower crop invariably showed PSND. Although marigold can serve as an efficient source of inoculum, this crop is grown only under irrigation on a limited scale.

Studies conducted on field-infected and mechanically inoculated plants of groundnut, sunflower and parthenium have so far failed to show seed transmission of the virus. However, these studies were limited to a small number of seeds and this aspect requires further investigation using large quantities of seed of more than one cultivar.

Conditions congenial for disease epidemics

The following conditions appear favorable for disease development.

- Pre-monsoon showers during late May or early June that encourage germination and growth of parthenium
- ii. Sowing groundnut during July by which time parthenium is in full bloom
- iii. Normal rains, promoting good growth of groundnut crop as well as parthenium accompanied with one or two dry spells that encourage thrips multiplication and movement eventually resulting in virus spread.

Disease Management

After the PSND epidemic in the rainy season of the year 2000 in Anantapur and adjoining districts, the disease incidence in subsequent years has remained very low. Several experiments conducted on the management of PSND remained inconclusive due to lack of high disease pressure. The empirical evidence suggests the following management practices that could help to contain the disease. However, these practices require onfarm validation under high disease pressure.

Resistant varieties

The most economical way to manage PSND, as for most other plant virus diseases, is to grow resistant varieties. But resistance to TSV has not yet been found in cultivated groundnut. In laboratory screening (mechanical inoculation using sap from virus-infected plant at 1:10 concentration), all 150 released varieties of groundnut in India were susceptible. Similarly, all 51 wild

Arachis accessions screened were positive for TSV in ELISA, except for one accession of A. chacoense (ICG 4983) which did not show symptoms inspite of TSV infection. However, upon screening with lower virus concentration (1:100 and 1:1000), a couple of improved varieties ICGV # 92267, 99029 and 01276 have shown consistently low disease incidence in repeated tests (ICRISAT, unpublished data).

Cultural practices

Removal of weed hosts

Removal of weeds, particularly parthenium germinated with early rains and growing wild in fallow lands, roadsides, and field bunds, is helpful in reducing PSND incidence.

Removal of infected groundnut plants from the field has no effect, as there is little likelihood of secondary spread of the disease within the groundnut crop.

Border cropping

During field surveys, soon after the PSND outbreak in Anantapur, natural barriers such as tall grass in the fields were found to protect the adjacent crops from the disease. In one case with tall grass barrier between a groundnut field and parthenium in the adjacent field, the crop was free from the disease whereas a nearby groundnut crop without barrier showed 70% PSND.

The naturally growing grass (2 m tall) might have obstructed the wind borne thrips and the inoculum carrying pollen of parthenium from landing on groundnut plants. Based on these observations, 7-11 rows of fast growing (tall) pearl millet, sorghum or maize as border crop around

groundnut fields were suggested to control the disease (Fig. 10).



Figure 10. Groundnut field with seven rows of maize as border crop.

Optimum plant population

Sub-optimum plant population leaves bare patches in the field which attract thrips landing. Optimum plant population, which gives quick ground cover, will discourage thrips landing on the groundnut crop.

Neighboring crops

It is advisable not to grow sunflower, marigold and other TSV susceptible crops adjacent to groundnut fields.

Chemical control

As is the case with most of the thrips-transmitted viruses, use of insecticides after the appearance of disease has no effect on disease control. However, preliminary observations from seed treatment trials conducted at Kadiri indicate low thrips damage and subsequent low PSND incidence with imidacloprid (Gaucho 70 WS) seed treatment. When conditions are congenial for disease occurrence (premonsoon showers), it would be advisable to treat the seed with imidaclorprid and follow it with regular systemic insecticide spray to control thrips in the early stages of crop growth.

Conclusions

Tobacco streak virus (TSV) caused the disease epidemic in groundnut crop in the rainy season of the year 2000 in Anantapur. This was the first record of TSV in groundnut and the disease was named as peanut stem necrosis disease (PSND). It is difficult to distinguish between PSND and PBND. another economically important virus disease of groundnut, based on symptoms alone. Techniques have been developed for precise diagnosis of the disease by ELISA and by the reaction of indicator hosts. TSV infects several economically important crop plants and survives on many weed hosts under field conditions. Parthenium, a widely distributed and symptomless carrier of TSV, plays a major role in perpetuation and spread of the disease. The role of infective pollen and flower-inhabiting thrips in the transmission of TSV has been established. Seed transmission of TSV in groundnut and other crop plants as well as in weed hosts requires further investigation. Various proposed disease management practices could not be validated on-farm due to lack of natural disease pressure in subsequent years. However, based on field observations and laboratory tests, interim measures such as the use of tolerant varieties, removal of weed hosts, border cropping, optimum plant population, seed treatment and use of systematic insecticides are suggested.

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