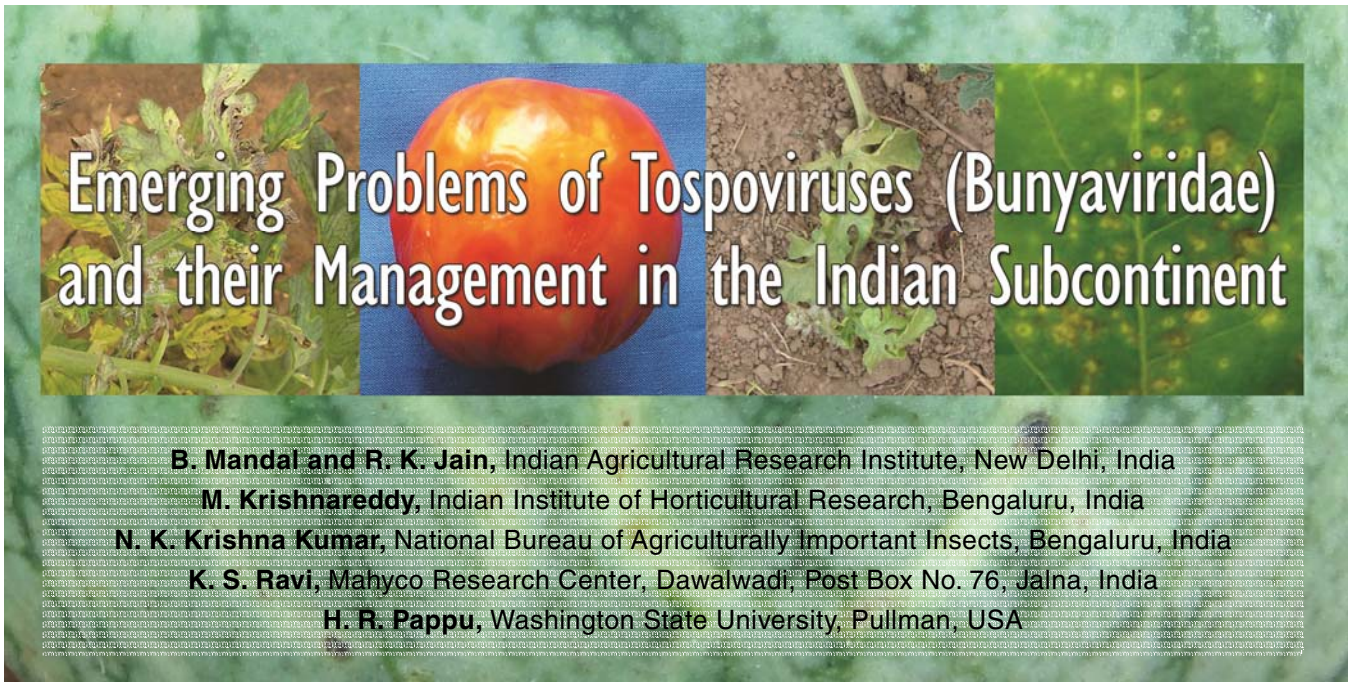

Emerging Problems of Tospoviruses (Bunyaviridae) and their Management in the Indian Subcontinent

B. Mandal and R. K. Jain, Indian Agricultural Research Institute, New Delhi, India
M. Krishnareddy, Indian Institute of Horticultural Research, Bengaluru, India
N. K. Krishna Kumar, National Bureau of Agriculturally Important Insects, Bengaluru, India
K. S. Ravi, Mahyco Research Center, Dawalwadi, Post Box No. 76, Jalna, India
H. R. Pappu, Washington State University, Pullman, USA

Tospoviruses (family *Bunyaviridae,* genus *Tospovirus*) are enveloped isometric RNA viruses with a tripartite genome containing small (S), medium (M), and large (L) segments of ssRNA. They are transmitted by thrips (Thysanoptera) in a propagative manner and are one of the most important plant virus groups infecting a wide range of economically important crop plants all over the world (63). Tospoviruses have emerged as serious viral pathogens affecting the cultivation of several field and horticultural crops (109). In the Indian subcontinent, due to their economic impact on a wide range of important crops, most of the research was carried out only in India, although tospoviral diseases are significant problems in several other countries in the subcontinent including Bangladesh, Nepal, Pakistan, and Sri Lanka. Several reviews are available on various aspects of tospoviruses and thrips vectors (7,21,36,63,64,106). The present review aims to highlight emerging disease problems, virus characteristics, vectors, diagnosis, epidemiology, and management of tospoviruses in the Indian subcontinent.

Historical Perspective

Disease symptoms similar to those induced by tospoviruses have been described in India since the 1960s on several crops such as blackgram (*Vigna mungo*), brinjal (*Solanum melongena*), chili (*Capsicum annuum*), cowpea (*Vigna unguiculata*), groundnut/peanut (*Arachis hypogaea*), mungbean (*Vigna radiata*), pea (*Pisum sativum*), potato (*Solanum tuberosum*), soybean (*Glycine max*), and tomato (*Solanum lycopersicum*) (6,11,23,39,44,61,62, 66–68,83,93). Since *Tospovirus* was a monotypic genus until 1990, with *Tomato spotted wilt virus* (TSWV) as the sole species, tospoviral diseases described in India were initially considered to be caused by TSWV. It was in 1992 that it was suggested based on serology that the bud necrosis disease of groundnut was caused by a tospovirus different from TSWV, and the virus was named *Groundnut bud necrosis virus* (GBNV) (80). GBNV was further confirmed as a distinct tospovirus based on nucleocapsid protein (N) gene sequence (90). Subsequently, another distinct tospovirus, *Peanut yellow spot virus* (PYSV), causing yellow spots on groundnut was identified (88). A new and unusual disease on watermelon characterized by leaf mottling and dieback of shoots was observed during 1991 and 1992 in parts of southern India (96), and a distinct tospovirus, *Watermelon bud necrosis virus* (WBNV), was found associated with this disease (34). The most recently reported tospoviruses in India include *Iris yellow spot virus* (IYSV) on onion (*Allium cepa*) (74) and garlic (*Allium sativum*) (20), and *Capsicum chlorosis virus* (CaCV) on tomato (48) and chili (*Capsicum annum*) (43).

Corresponding author: R. K. Jain, Advanced Centre for Plant Virology, Division of Plant Pathology, Indian Agricultural Research Institute, New Delhi, India; E-mail: rakeshjain56@yahoo.co.in

http://dx.doi.org/10.1094/PDIS-06-11-0520

Geographic Distribution and Economic Impact

The geographic distribution of tospoviruses in the Indian subcontinent varies (Fig. 1). In India, GBNV and WBNV are widely distributed and endemic in many states including Andhra Pradesh, Gujarat, Haryana, Himachal Pradesh, Karnataka, Kerala, Madhya Pradesh, Maharashtra, Orissa, Punjab, Rajasthan, Tamil Nadu, Uttar Pradesh, and West Bengal. IYSV has been found in Gujarat, Karnataka, Madhya Pradesh, Maharashtra, Tamil Nadu, and Uttar Pradesh. CaCV is present in northern, central, and southern parts of India (43,49). PYSV has been reported from Andhra Pradesh while its presence in other states cannot be ruled out. The northeastern states of India are not known to be affected by any tospovirus. In Bangladesh, Nepal, Pakistan, and Sri Lanka, bud necrosis disease of groundnut, tomato, and watermelon is known but the causal viruses were not conclusively identified (1,10,15,17,92).

GBNV is currently recognized as the most economically important tospovirus, as losses due to GBNV alone have been estimated at more than US$89 million per annum in Asia (79). GBNV caused 70 to 90% loss of groundnut in India (94). Serious outbreak of GBNV was reported in different tomato growing regions in Maharashtra, Karnataka, and Andhra Pradesh, where up to 100% disease incidence was recorded during 2003 to 2006 (47). Besides groundnut and tomato, losses up to 29% have been recorded in potato due to stem necrosis disease caused by GBNV (95). Cultivation of watermelon was seriously affected due to an outbreak of WBNV in parts of southern India, forcing farmers to abandon growing watermelon due to total crop loss (96). The impact of IYSV and CaCV on onion and tomato, respectively, in the country is not known, although yield reduction was anticipated due to se-

vere and widespread incidence of the disease in several parts of Maharashtra (49). In Bangladesh, spotted wilt or bud necrosis disease of tomato has emerged as a significant problem, and most of the tomato cultivars are susceptible (17). During 2010, an epidemic of tospovirus seriously affected cultivation of tomato in the Rajshahi district of Bangladesh (Shamim Akhter, Bangladesh Agricultural Research Institute, *personal communication*). In Pakistan, bud necrosis in groundnut is not a serious problem; however, disease incidence up to 15% has been recorded (15).

Diseases and Virus Characterization

More than 20 tospoviruses have been reported from all over the world (63). To date, five tospoviruses, CaCV, GBNV, IYSV, PYSV, and WBNV (Table 1), are known in India, and any of the known tospoviruses is yet to be characterized from other countries of the Indian subcontinent.

Groundnut bud necrosis virus. The field symptoms of GBNV in groundnut are very well described (Fig. 2A to C). Initially, mild chlorotic spots appear on young quadrifoliate leaves, and subsequently necrosis and chlorotic rings develop. In rainy and post-rainy seasons, necrosis of terminal bud is the main characteristic symptom. Secondary symptoms such as stunting, axillary shoot proliferation, and malformation of leaflets are common. Plants infected early are bushy, stunted, and die prematurely. If plants older than one month are infected, the symptoms are restricted to a few branches only (82).

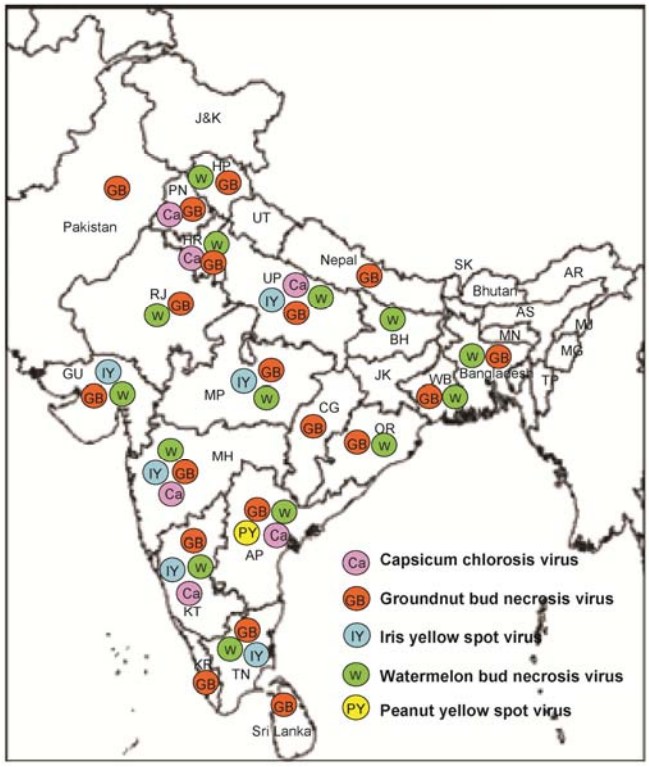

Fig. 1. Distribution of tospoviruses in the Indian subcontinent. HP: Himachal Pradesh, PN: Punjab, HR: Haryana, UP: Uttar Pradesh, RJ: Rajasthan, MP: Madhya Pradesh, BH: Bihar, WB: West Bengal, OR: Orissa, CG: Chhattisgarh, MH: Maharashtra, GU: Gujarat, AP: Andhra Pradesh, KT: Karnataka, TN: Tamil Nadu, KR: Kerala. Tospoviruses have not been examined in the following states of India: J&K: Jammu and Kashmir, UT: Uttaranchal, JK: Jharkhand, SK: Sikkim, AS: Assam, AR: Arunachal Pradesh, MG: Meghalaya, NL: Nagaland, MN: Manipur, MJ: Mizoram, TP: Tripura. Tospoviruses in Bangladesh, Pakistan, Nepal, and Sri Lanka are suspected largely based on occurrence of disease.

GBNV is the most widespread of all the tospoviruses known in India. In addition to groundnut, important crops such as cowpea, mungbean, pea, potato, soybean, and tomato are known to be affected by GBNV (Fig. 2D to F) (2,4,8,11,32,71,104,108). The experimental host range of GBNV includes several species belonging to the families Amaranthaceae, Asteraceae, Chenopodiaceae, Cucurbitaceae, Fabaceae, Malvaceae, and Solanaceae (70,86,104).

The complete genome of GBNV (type isolate, groundnut) has been sequenced, which consisted of three linear single-stranded RNA molecules, the L (8.9 kb), the M (4.8 kb), and the S (3.05 kb) RNAs (27,90,91). The L RNA is entirely of negative polarity, with one open reading frame (ORF) located on the viral complementary strand encoding the viral polymerase of 330 kDa L-protein (2,877 amino acids). The L-protein had the highest identity in the core-polymerase domain with the corresponding regions of the TSWV and INSV (27). The M RNA of GBNV encodes a 34.3 kDa movement protein (NSm) in the viral sense and a 127.3 kDa precursor to the two viral membrane glycoproteins, Gn and Gc, in the viral complementary sense (91). Recently, the genome organization of M-RNA from GBNV (mungbean isolate) was determined (87) and compared with the type isolate (91). Comparative sequence analysis of GBNV isolates from groundnut, mungbean, and tomato revealed that the genome of the M RNA was considerably different in their intergenic regions (56 to 89% sequence identity) and Gn/Gc protein. The topology of Gn/Gc revealed the presence of both *N*-glycosylation and *O*-glycosylation sites in GBNV-mungbean (Mb) isolate, whereas only *N*-glycosylation sites were present in the GBNV-type isolate. The S RNA of GBNV encodes 49.5 kDa nonstructural protein (NSs) in the virus sense and the 30.6 kDa N protein in the virus complementary sense (90). The NSs protein is expressed from a virus sense 1,320 nt subgenomic RNA and the N protein from a 831 nt virus complementary sense subgenomic mRNA. The complete genome of S-RNA of GBNV-Mb was sequenced and found very similar to that of the type isolate (87). The NSs protein of GBNV has been characterized as a bifunctional enzyme containing RNA stimulated ATPase and 5′ phospatase activities, which possibly participates in suppression of the host defense mechanism (54).

Watermelon bud necrosis virus. The field symptoms of WBNV in watermelon initially develop as chlorotic mottling, yellow spots or patches, and mild crinkling of leaves (Fig. 3A). Subsequently, necrosis of buds in the growing tips results in dieback of vines (Fig. 3B). In the young crop, rapid dieback and wilting of plants develop dramatically causing a total loss in the affected plants. In the mature crop, shortened internodes, upright growth of younger shoots, necrosis on stem, petiole, and fruit stalk are commonly seen. Infected plants produce unmarketable small, deformed fruits with uneven surface, and necrotic or chlorotic rings, depending on the cultivar (Fig. 3C and D). GBNV is predominant in leguminous and solanaceous hosts, while WBNV is largely confined to cu-

Table 1. Tospoviruses recorded in India

Virus	Acronym	Record of emergence				Reference
		Year	Crop	Place	Key symptoms	
Capsicum chlorosis virus	CaCV	2006	Chili	Karnataka	Apical necrosis	43
Groundnut bud necrosis virus	GBNV	1968	Groundnut	Andhra Pradesh	Bud necrosis	80
Irish yellow spot virus	IYSV	2002-03	Onion	Maharashtra	Chlorotic lesions	74
Peanut yellow spot virus	PYSV	1991	Groundnut	Andhra Pradesh	Yellow spot	88
Watermelon bud necrosis virus	WBNV	1991-92	Watermelon	Karnataka	Bud necrosis	34

curbitaceous hosts such as ridge gourd and cucumber (31,56). Recently, WBNV was detected in chili and tomato in northern India (47). Under experimental conditions, WBNV is sap transmissible to different plant species in Solanaceae and Cucurbitaceae (96).

The complete genome of WBNV has recently been characterized, confirming its status as a distinct tospovirus species affecting cucurbits in India (46,47,52). The S, M, and L segments of WBNV are 3.4, 4.7, and 8.9 kb long, respectively, and possess the typical

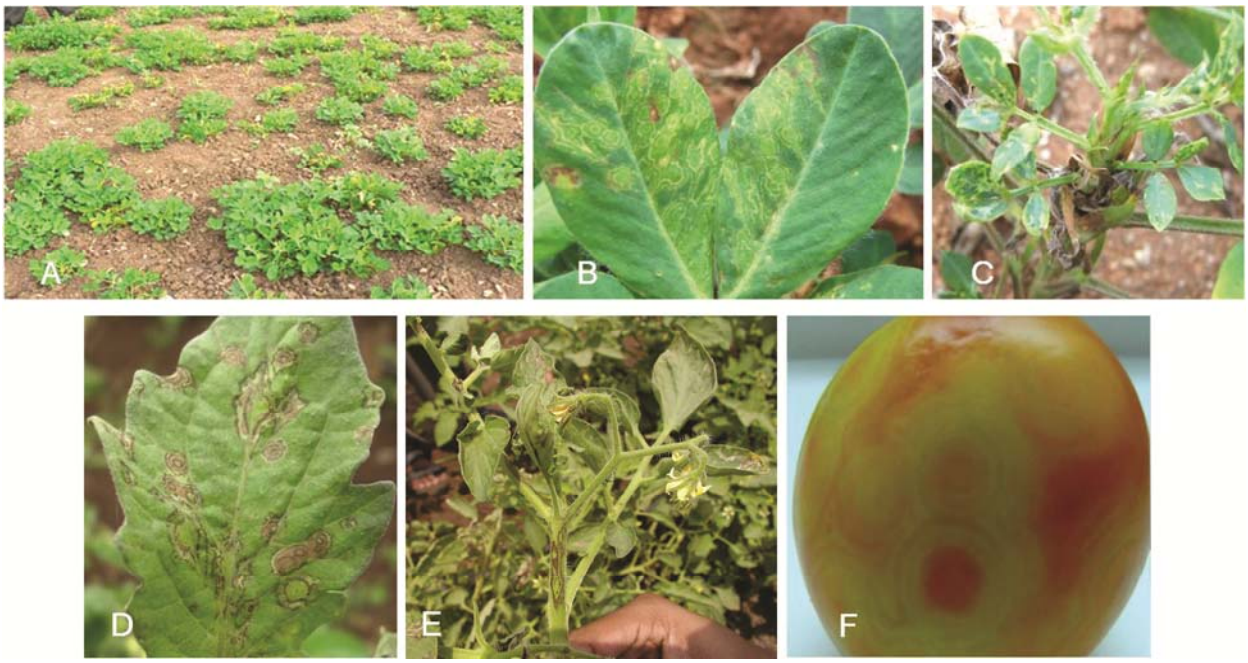

Fig. 2. *Groundnut bud necrosis virus* on groundnut **(A to C)** and tomato **(D to F)**. **A,** Field outbreak of bud necrosis disease, **B,** chlorotic rings on leaves, **C,** stunting of plant with mosaic mottling and bud necrosis symptoms on groundnut. **D,** Necrotic rings on leaf, **E,** stem necrosis, **F,** concentric rings and patchy color on fruit of tomato.

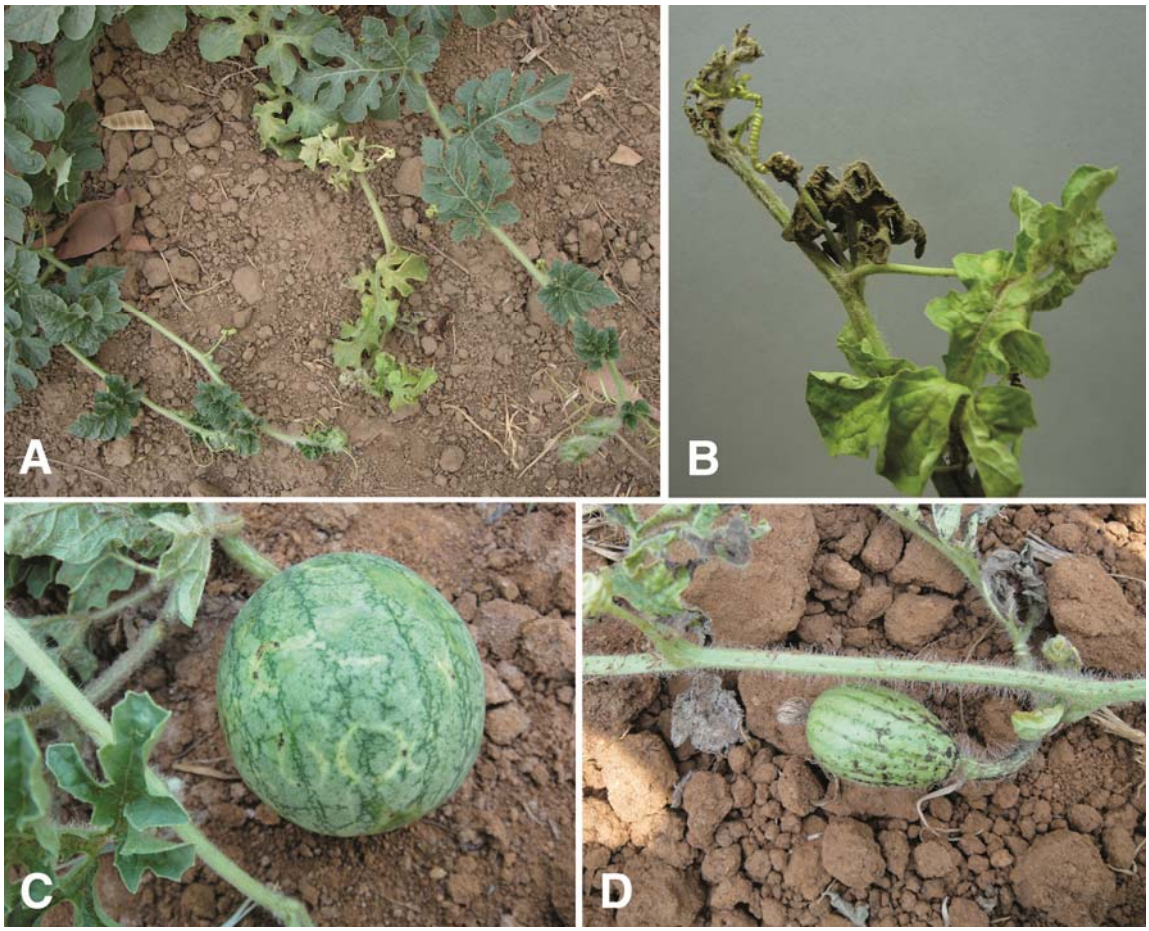

Fig. 3. *Watermelon bud necrosis virus* on watermelon: **A,** chlorosis and dieback of shoot, **B,** bud and shoot necrosis, **C,** chlorotic ring on fruit, and **D,** necrosis on developing fruit.

features of tospovirus genome organization. Phylogenetic analysis based on complete M RNA sequence revealed three distinct clusters of tospoviruses with respect to WBNV: (i) the close relatives, CaCV, GBNV, Gloxinia tospovirus-HT1, and *Watermelon silver mottle virus* (WSMoV) sharing 73.9 to 79.1% sequence identity; (ii) the intermediate relatives, IYSV, *Melon yellow spot virus* (MYSV), and *Tomato zonate spot virus* (TZSV) sharing 62.0 to 65.3% sequence identity; and (iii) the distant relatives, *Impatiens necrotic spot virus* (INSV) and TSWV sharing only 46.3 and 44.7% sequence identity, respectively. Among these tospoviruses, GBNV was closest to WBNV sharing 79.1% sequence identity. The melon-infecting tospoviruses, MYSV and WSMoV, known to occur in Japan and Taiwan (38,53), shared only 63.3 and 75.2% identity, respectively, with WBNV.

Peanut yellow spot virus. PYSV was identified as a distinct tospovirus in groundnut based on thrips transmission, host range, and serology (81). The disease caused by PYSV in groundnut is characterized by yellow spots followed by necrosis on leaves. Incidence of PYSV up to 90% has been observed in southern India, but impact on yield loss is not known. PYSV is serologically distinguishable from TSWV, INSV, and GBNV (89). PYSV was suggested as a new species based on S RNA sequence characteristics (88). The N and NSs proteins of PYSV are significantly different from those of the other tospoviruses, as they shared low sequence identity of 24 to 28% and 16 to 21% with the corresponding genes of known tospoviruses.

Capsicum chlorosis virus. CaCV is recently reported to be prevalent in northern, central, and southern parts of India. Symptoms of CaCV in tomato and chili (Fig. 4A to D) are very similar to those induced by GBNV. The disease symptoms in chili are yellow spots or patches followed by occasional chlorotic concentric rings on leaves. In tomato, bud necrosis results in dieback of shoot. Fruits of tomato display uneven ripening and chlorotic concentric rings. CaCV was first reported from Australia and differs from the Indian isolate by host range (37,49). CaCV has a similar host range to that of GBNV.

The complete sequences of S, M, and L segments of CaCV genome have recently been reported (49). The intergenic region of S RNA of an Indian isolate of CaCV is considerably shorter than CaCV-Thailand isolate and therefore shared very low sequence identity (39.4%). Interestingly, based on the intergenic region sequence, CaCV occurring in India is closer to GBNV (76%) compared to the CaCV-Thailand isolate.

Iris yellow spot virus. IYSV is an important constraint in onion and garlic in several countries (63). In India, IYSV was recently identified in both onion and garlic (20,74). IYSV shows a wide range of symptoms. In onion, disease appears as yellow stripes, spindle-shaped chlorotic lesions, or rings. In garlic, straw-colored spindle-shaped spots coalesce to form a large patch on the leaves. The virus appears to be more prevalent in onion. Sequence characteristics of the N gene of several IYSV isolates have been recently reported (47).

Diversity of Tospoviruses

Biological diversity. GBNV, which is primarily known to affect groundnut, now has been identified to cause necrosis disease in diverse crops: blackgram, brinjal, chili, cowpea, mungbean, pea, potato, and soybean. Comparison of disease response of GBNV isolates GN, MB, Tom-1, Tom-2, and P, originating from different hosts and locations in northern and southern India, revealed limited biological diversity (Table 2). These isolates induced chlorotic or necrotic local lesions followed by systemic necrosis in different crop hosts. The isolates, irrespective of their origin, induced identical disease responses in groundnut and mungbean and showed

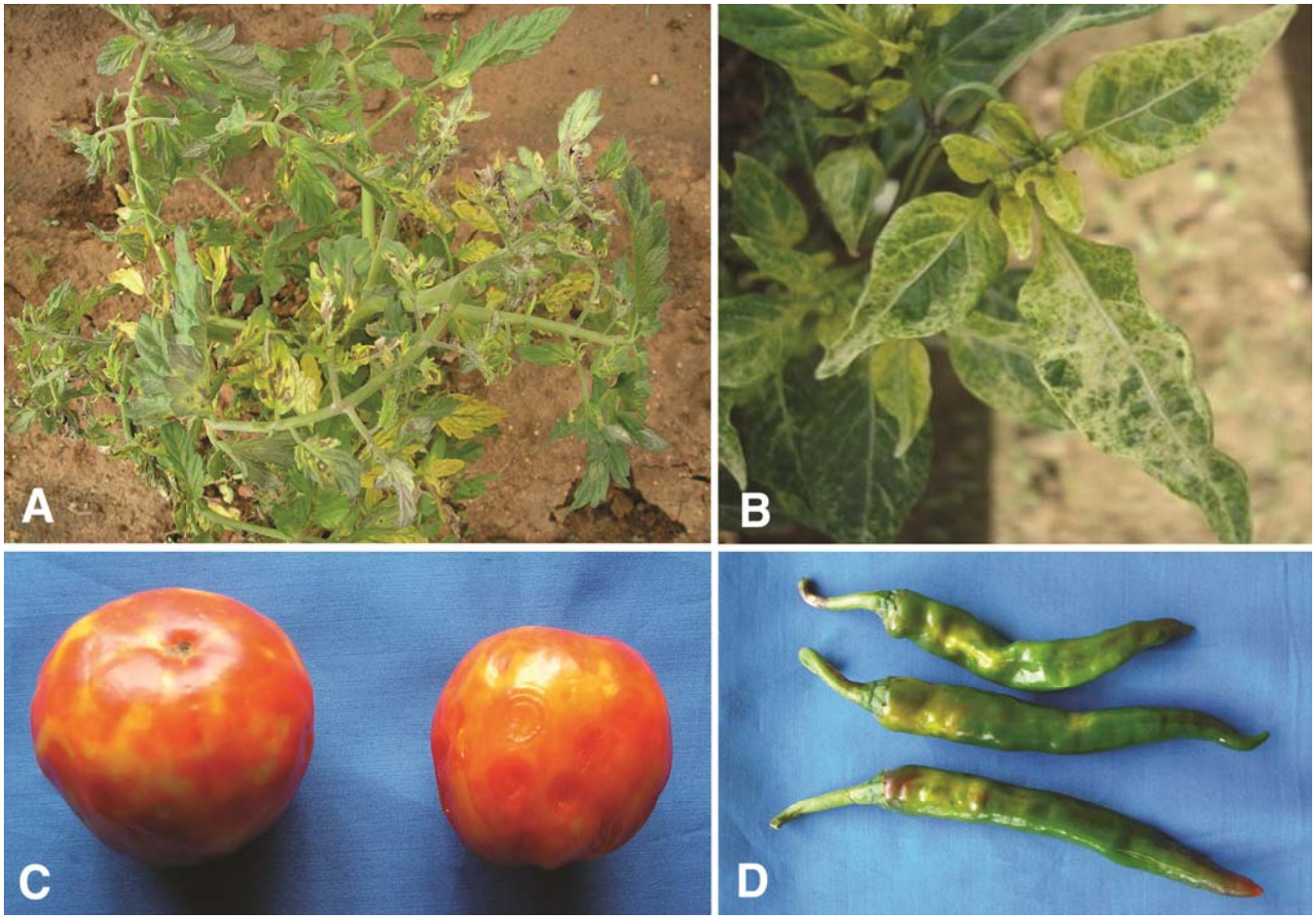

Fig. 4. *Capsicum chlorosis virus* on tomato and chili: **A,** necrosis on tomato leaves and **C,** uneven ripening of tomato fruits. Chlorosis on **(B)** leaves and **(D)** fruits of chili.

some variations in cowpea, soybean, tomato, and cucumber. The host-reactions of Mb isolate originating from mungbean in Delhi was very similar with the type isolate-GN originating from groundnut in Hyderabad in most of the crop hosts except in soybean, where Mb isolate failed to induce systemic necrosis (104). Two tomato isolates of GBNV, Tom-1 isolated from Kerala and Tom-2 from Hyderabad, differed in their host-reactions in cowpea and soybean. Biological diversities of GBNV isolates infecting potato, chili, brinjal, and soybean are not known. The host-reactions of CaCV isolates from tomato and chili have been compared and no difference was observed (49).

Genetic diversity. Genetic diversity studies have been largely based on the N gene, followed by the NSm gene. Amino acid sequence identity (>90%) of N gene is considered to be one of the important criteria for species demarcation in tospoviruses (24), and N gene sequences of more than 100 GBNV isolates originating from different hosts and locations are available in GenBank. Comparison of amino acid sequences of these isolates revealed divergence of up to 8.4%, and the isolates were classified into eight different evolutionary clusters irrespective of their geographical origin or host (Fig. 5). GBNV isolates from mungbean, tomato, and cowpea from Kerala, Uttar Padesh, and Delhi were nearly identical (99% identity) to the GBNV type isolate (35,104). GBNV isolates from tomato from Kerala, Maharashtra, Tamil Nadu, and Uttar Pradesh showed 96 to 100% identity with type isolate of GBNV (71,108). GBNV from potato causing stem necrosis disease in Gujarat, Madhya Pradesh, and Rajasthan showed 98 to 99% identity with the GBNV type isolate (32). Comparison of NSm

Table 2. Host reactions to *Groundnut bud necrosis virus* isolates originating from different hosts and locations in India

		Host reaction[a]						
Isolates	**Origin**	**Groundnut**	**Mungbean**	**Cowpea**	**Soybean**	**Tomato**	**Cucumber**	**Reference**
Groundnut (GN)	Andhra Pradesh	S+	S+	S+	S+	S+	L+	23
Mungbean (MB)	Delhi	S+	S+	S+	L+	NT	L+	104
Tomato (Tom-1) K	Kerala	S+	S+	S+	O	S+	O	108
Tomato (Tom-2)	Andhra Pradesh	S+	S+	L+	S+	S+	NT	67
Pea (Pea)	Andhra Pradesh	S+	S+	S+	S+	S+	NT	68

[a] O: No infection; S+: systemic necrosis; L+: chlorotic/necrotic local lesion; NT: not tested.

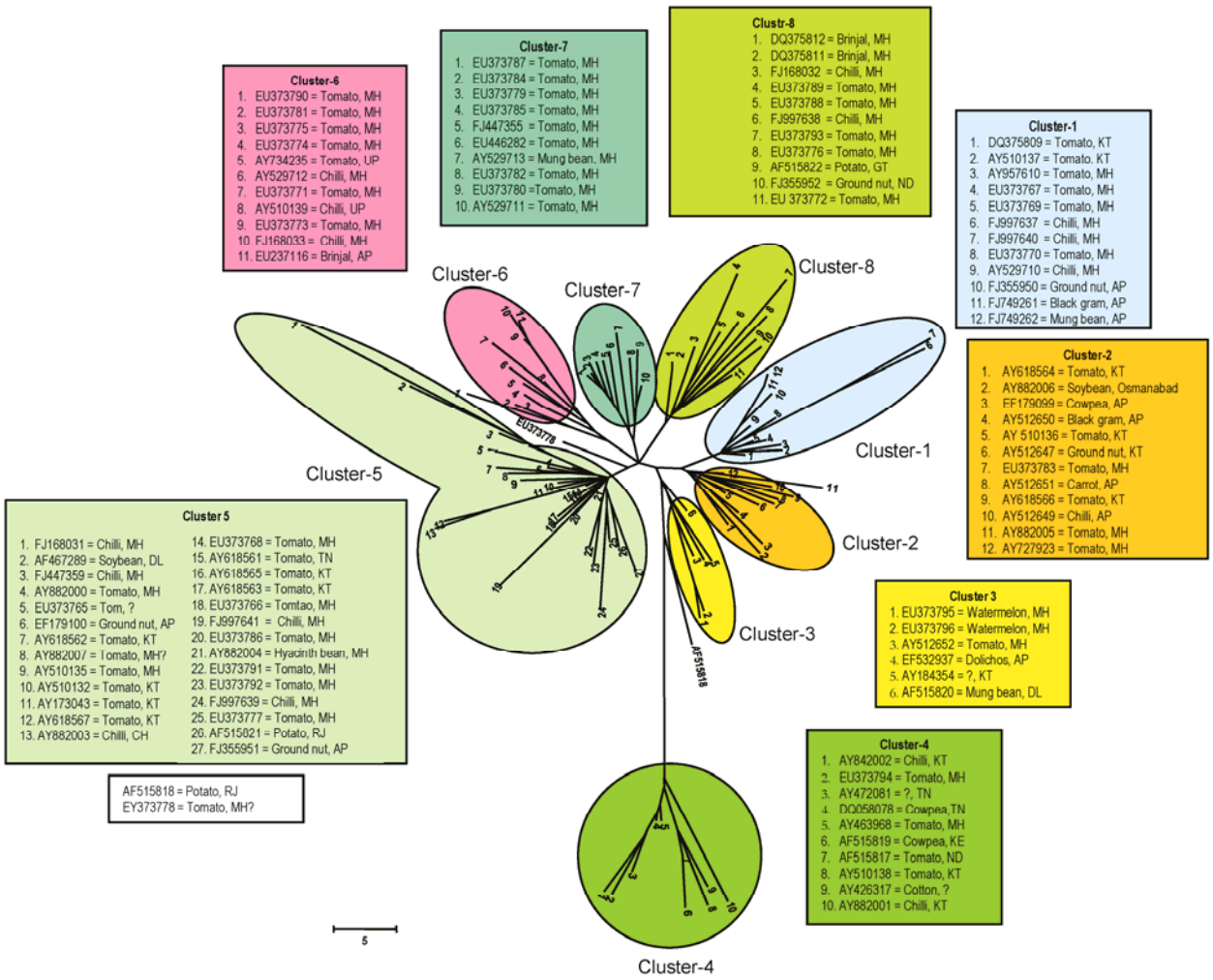

Fig. 5. Phylogenetic clustering of isolates of *Groundnut bud necrosis virus* from diverse crops and places in India. Maximum Parsimony tree was constructed in MEGA4 sofware (http://www.megasoftware.net).

gene also revealed limited diversity (2 to 3%) in GBNV isolates from cowpea, groundnut, tomato, and potato from Kerala, Madhya Pradesh, Rajasthan, Tamil Nadu, and Uttar Pradesh (2,3,71).

Genetic diversity in IYSV, CaCV, and WBNV occurring in India has been described based on fewer isolates compared to GBNV (47). Diversity in PYSV, however, has not been studied so far. N gene sequence of 31 IYSV isolates from onion showed divergence of up to 5.2% at the amino acid level. WBNV is a serious problem in watermelon, and 12 isolates that were sequenced showed 7.0 and 8.55% divergence based on the amino acid sequences of N and NSm proteins, respectively.

Thrips as Vectors

Tospoviruses are exclusively vectored by thrips in a circulative and propagative manner (36,84,113). In the thrips body, tospovirus is transported through midgut and salivary epithelium membrane barriers to salivary gland or through ligaments connecting midgut and salivary glands (58). Of 11 species of thrips recorded as vectors of tospoviruses throughout the world (63), five thrips vectors, *Ceratothripoides claratris, Frankliniella schultzei, Scirtothrips dorsalis, Thrips palmi,* and *T. tabaci,* have been reported from India. The distribution of thrips genera in the vegetable crops surveyed during 2006 to 2007 in Andhra Pradesh, Karnataka, and Maharashtra showed a dominance of *Thrips* sp. in onion, *Scirtothrips* sp. in chili, and *Frankliniella* sp. in tomato (13). Inappropriate identification perhaps resulted in *Thrips flavus* being reported as a vector of WBNV (96). Not much is known about the virus–vector relationships of thrips-transmitted tospoviruses in the Indian subcontinent and is a fertile ground for further research.

T. palmi is a suspected vector for GBNV (50) and WBNV (96) in India. It has been recorded as a pest of *Capsicum annuum, Citrullus lanatus, Cucumis melo, Cucumis sativus, Cucurbita* spp., *Glycine max, Gossypium* spp., *Helianthus annuus, Nicotiana tabacum, Phaseolus vulgaris, Pisum sativum, Sesamum indicum, Solanum melongena, Solanum tuberosum,* and *Vigna unguiculata.* In glasshouses, economically important hosts of *T. palmi* are *Capsicum annuum, Chrysanthemum* spp., *Cucumis sativus, Cyclamen* spp., *Ficus* spp., and *Solanum melongena.* Meena et al. (57) reported *S. dorsalis* as a vector of GBNV in tomato. While *T. tabaci* has been identified as a vector of IYSV in Israel (22) and *C. claratris* as a vector of CaCV in Thailand (69), the vector status of these thrips species in relation to IYSV and CaCV has yet to be established. There is also no data on the biotype composition of the major thrips species such as *T. tabaci* and *T. palmi*. PYSV was efficiently transmitted by *S. dorsalis* in groundnut. Larvae could acquire the virus in 30 min, and the maximum percent transmission of 43.8% by individual insects resulted following two days acquisition access period. Single adult thrips transmitted the virus after minimum inoculation access period (IAP) of 30 min. The percent transmission increased with an increase in IAP (26). While there is no record of western flower thrips (*Frankliniella occidentalis*) in the subcontinent, strategies to contain this vector and the tospoviruses that can potentially be transmitted need to be developed in the event this important thrips vector is introduced into the region.

Epidemiology of Tospoviruses

Since tospoviruses are not seedborne, it is assumed that the primary spread of tospoviruses is by thrips coming from other crops or weeds, whereas secondary spread takes place from infected plants within a field. The primary sources of GBNV include a range of solanaceous and fabaceous hosts such as blackgram, cowpea, eggplant, groundnut, mungbean, pepper, potato, soybean, and tomato, which can sustain virus infection and support thrips vector multiplication (78). Bud necrosis disease of groundnut is mostly monocyclic type, and disease incidence depends on infection by viruliferous thrips that acquire the virus from other crops or alternate hosts. *Ageratum conyzoides* has been shown to support GBNV and vector multiplication (77). In another study, spread of WBNV was influenced by the incidence of thrips population, maximum temperature and relative humidity (during morning hours). While minimum temperature negatively influenced WBNV incidence, wind velocity and rainfall failed to influence the WBNV spread or thrips population build-up. The rate of spread of WBNV was highest in watermelon cultivars Arka Manik, followed by NS 295 and Madhu Bala (42,45).

Usually, raising melons early (October to February) in the season is advocated in southern India to minimize WBNV, as it coincides with fewer thrips. WBNV can be devastating even during monsoon (July to October) if there is a dry spell of 25 to 30 days. Thrips need 10 to 12 days from egg to adult development, and a dry spell in the midst of monsoon can facilitate large-scale multiplication and subsequent migration leading to a WBNV outbreak (M. Krishnareddy, *unpublished data*).

Diagnosis of Tospoviruses

Tospovirus diagnostics received considerable attention during the last two decades in India. This has led to the identification of various tospovirus species. A combination of bio-, immuno-, and nucleo-based assays have been developed. For GBNV, WBNV, and CaCV, cowpea (*Vigna unguiculata* 'Pusa Komal' and 'C-152') has been identified as a suitable indicator host, which produces localized as well as occasional systemic symptoms (23,31,49,71,96). Local symptoms included mild chlorotic spots, which turned necrotic with necrotic rings followed by yellowing and wilting of inoculated leaves, and systemic symptoms included necrotic spots or veinal necrosis on young leaves and necrosis of stem (Fig. 6D to F). *Nicotiana benthamiana* is another important indicator, as well as propagative host, which produces systemic chlorotic mottling, necrosis, and rapid wilting (Fig. 6A to C).

Polyclonal antibodies (PAb) to N protein of WSMoV and GBNV were capable of detecting GBNV, WBNV, and CaCV originating from different hosts and locations by enzyme-linked immunosorbent assay (ELISA) (8,31,35), suggesting these tospoviruses are serologically indistinguishable. On the basis of nucleocapsid protein (N) serology, GBNV, WBNV, and CaCV were grouped in WSMoV serogroup, IYSV in IYSV serogroup, and PYSV in unclassified serogroup (18). As purification of tospovirus is difficult, recombinant N protein expressed in *Escherichia coli* has been utilized for the production of PAb (33), which led to commercialization of an ELISA-based diagnostic kit for GBNV and other serologically related tospoviruses in India (55). Monoclonal antibodies developed against N protein of GBNV were highly specific and capable of differentiating GBNV and WBNV isolates, and a simple dot blot assay was developed for detection of GBNV in field samples (30).

Unequivocal identification of tospovirus species is based on N-gene sequence with a threshold level of <90% amino acid sequence identity (24). Reverse transcription–polymerase chain reaction (RT-PCR) using virus-specific N-gene primers has been standardized and validated (34,71). Specific primers and RT-PCR methods were successfully used for the detection of GBNV and WBNV, which are otherwise serologically indistinguishable (B. Mandal, *unpublished results*). Further, a single tube one-step RT-PCR method was developed using degenerate, conserved forward and virus-specific reverse primers for the specific detection of GBNV, WBNV, and CaCV (49).

Management of Thrips and Tospoviruses

Management of tospoviruses is difficult because of their wide host range, thrips resistance to insecticides, and lack of durable resistance in crop hosts. Control measures for tospoviruses, which include phytosanitary, cultural resistance, host plant resistance, and chemical and biocontrol measures, need to be based on sound epidemiological principles such as internal and external sources of inoculum, early or late phases of virus spread, and vulnerable stages in the virus/vector/crop cycle (36).

Cultural practices. Agronomic practices followed for crop cultivation strongly influence build-up of thrips population and tospovirus infection. Tospovirus infection can be reduced by sowing the crop when the most sensitive stage is least invaded by thrips

vector. Adjusting sowing date so as to avoid thrips vectors markedly influences disease build-up in groundnut and other crops. Reddy et al. (78) showed that peak thrips flight activity of *F. schultzei* near Hyderabad in peninsular India occurred from late July to early September and late November to mid-January. Sowing of groundnut during this period resulted in higher incidence of GBNV and subsequent losses to crop production. In southern India, Reddy et al. (79) observed that groundnut crops sown early with the onset of rains (mid to late June) escaped GBNV infection, as the thrips vector flights usually occurred in July and August. In contrast, Thira et al. (105) observed maximum GBNV infection in a groundnut crop sown during May in northern India. Sowing of mungbean during the second half of May to the first half of June in summer and late sowing in spring helped in containing the thrips infestation as well as GBNV infection (99). Potato planted on 13 November completely escaped stem necrosis disease in central India (98). Under experimental conditions, the number of groundnut plants affected per unit area remained the same as plant density increased (78). Further, close spacing was effective for the groundnut crop in peninsular and central India as increasing plant density compensated yield loss due to bud necrosis disease. Roguing of symptomatic tomato seedlings during and soon after transplanting is a simple and effective measure in reducing virus incidence and crop loss without incurring extra costs for spraying pesticides to control thrips vectors (60).

Physical barriers that limit the movement of thrips have been shown to reduce GBNV incidence (79). Intercropping of groundnut with maize (*Zea mays*), pearl millet (*Pennisetum typhoides*), pigeonpea (*Cajanus cajan*), and sorghum (*Sorghum vulgare*) (3:1) significantly reduced GBNV incidence. Similarly, intercropping of mungbean with pearl millet (2:1) was best in reducing GBNV infection (100). Castor and sunflower were not effective in reducing the incidence of this disease and increasing the pod yield of groundnut (29). In watermelon, border cropping with two rows of maize significantly reduced WBNV incidence, and there was a delay of 10 to 15 days in the initial incidence.

The role of plastic mulches in reducing tospovirus incidence in commercial fields of tomato, pepper, muskmelon, and watermelon was recently tested. In tomato and pepper, black plastic mulch was very effective in reducing GBNV incidence up to 11% and thrips population up to 8.5%. Aluminum-surfaced plastic mulch gave significant reduction in WBNV incidence in cucurbit crops and was superior to black plastic mulch. Disease incidence in the mulched treatments could be correlated with vector thrips populations and increase in yield (M. Krishnareddy, *unpublished results*).

Thrips management by chemical measures. Thrips-transmitted viruses are rarely managed by using pesticides, as virus acquisition and transmission are closely linked to the developmental stage of the insect and only a small number of thrips vectors can result in a high rate of virus spread (107,113). Many thrips species are also resistant to insecticides, and their population may not be effectively controlled by chemical measures. Furthermore, tospovirus epidemics are commonly caused by dispersing thrips that are transient on the crop and insecticides are less likely to control this population. However, insecticides still constitute an important tactic in managing tospovirus diseases. High and frequent doses (400 g a.i. ha^{-1} at 3- or 5-day intervals) of dimethoate resulted in reduction of bud necrosis in groundnut in India, whereas low rates with longer intervals of dimethoate application (100 g a.i. ha^{-1} at 7- or 10-day intervals) induced higher levels of bud necrosis disease inci-

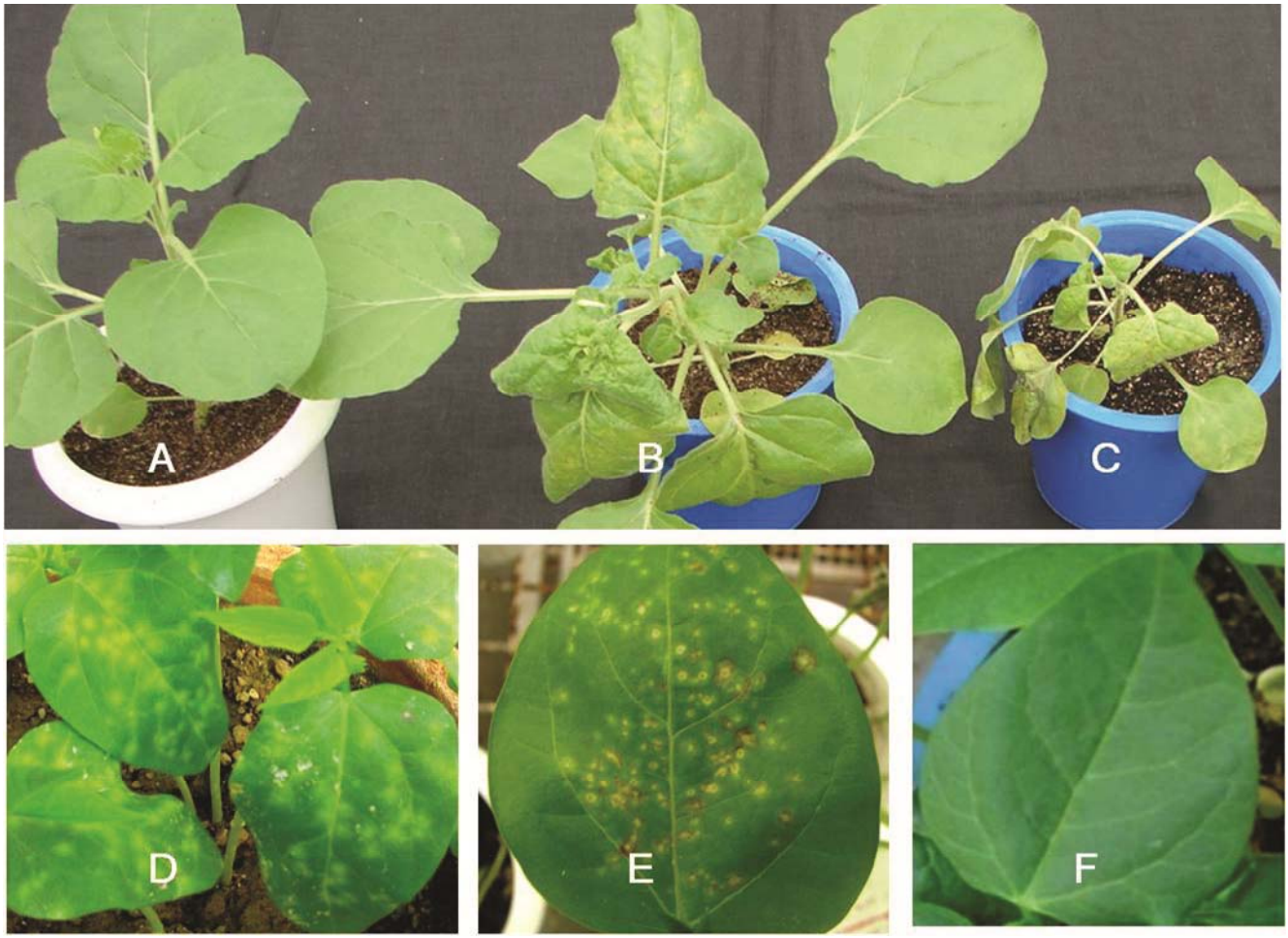

Fig. 6. Bioassay of *Groundnut bud necrosis virus*. **A to C,** *Nicotiana benthamiana*, a systemic and propagative host: **A,** healthy plant, **B,** systemic mottling, and **C,** wilting of shoots and top leaves following sap inoculation. **D to F,** *Vigna unguiculata* 'Pusa Komal', a local lesion assay host: **D,** chlorotic spots, **E,** chlorotic spots turn into necrotic lesions, and **F,** healthy leaf.

dence (114). Potato seed tuber dip in 0.07% imidacloprid plus one spray of 0.07% imidacloprid at 21 days or two foliar sprays of 0.07% imidacloprid at 21 to 35 days postplanting were effective in reducing potato stem necrosis disease caused by GBNV (98). Various application schedules of imidacloprid, thiamethoxam, acetamiprid, fipronil, dimethoate, fenvalerate, and azadirachtin were effective in managing *T. palmi* population in mungbean. Imidacloprid resulted in the most satisfactory control of *T. palmi* as well as the GBNV incidence in mungbean (101). Spraying of Acephate (Phosphorus Pentasulfide) or Regent (Fipronil) in combination with neem oil at 10 days interval from crop emergence to fruit formation was effective in reducing the thrips population and minimizing losses due to WBNV in watermelon (9).

Thrips management by biological measures. Exploiting natural enemies and predators of thrips to manage vector populations has been the subject of many studies (19). *T. tabaci* on onion is attacked by a eulophid parasitoid, *Ceranisus* sp., but its level of parasitism was low (up to 2 to 3%) under open field conditions. Another eulophid, *Thripobius* sp., accounted for more than 60% parasitism of *S. dorsalis* on capsicum under polyhouse conditions. However, occurrence of a few predatory anthacorid bugs, *Orius maxidentex* (Ghauri) and *O. tantillus* (Motschulsky), was observed on *S. dorsalis* on tomato. A study on the feeding potential of *O. tantillus* revealed that the predator was capable of preying on 43 to 336 thrips (with a mean of 166 thrips) during its adult period of 34 days (A. Krishnamoorthy, *personal communication*). A mass production technology has been developed for *O. tantillus* using thrips, flower buds, and French bean as sites of oviposition. *S. dorsalis* was effectively kept under control on capsicum by releasing *O. tantillus* predator (two per plant) under polyhouse conditions (41). An unidentified phytoseiid predatory mite of the genus *Euseilus* sp. was very effective in preying on onion thrips under laboratory conditions. Each predator consumed a mean of 16.5 second instar larvae per day (A. Krishnamoorthy, *personal communication*). Among several entomopathogens such as *Verticillium lecanii*, *Metarhizium anisopliae* var. *anisoliae*, *Beauveria bassiana,* and *B. brongniertii* screened for their efficacy against chili thrips, it was observed that *M. anisopliae* isolate obtained from thrips was effective in controlling thrips under field conditions; however, pesticides and antagonists are likely to interfere with the potential of entomopathogens (40).

Host Resistance

Resistance in groundnut. Evaluation of germplasm and breeding lines has resulted in identification of sources of resistance to GBNV in groundnut (51,79). Robut 33-1, a cultivar commonly grown by small farmers in Asia and Africa, has a marked degree of resistance to *F. schultzei* and some tolerance to GBNV infection (5). Other genotypes with higher levels of thrips resistance than Robut 33-1 have been crossed with high-yielding lines. Thus the field resistance in some of the groundnut genotypes may be due to resistance to the vectors combined with tolerance to GBNV.

Ramapandu and Raychaudhuri (73) screened 320 groundnut varieties under natural conditions; 56 varieties were free from bud necrosis and 15 were assessed as moderately resistant. When these 71 varieties were further tested in the greenhouse using graft inoculation, only three genotypes, EAH1010, EAH1232, and TMV7, were considered resistant. Desai (16) studied the response of 137 groundnut genotypes to GBNV during summer 1993–1994 in Karnataka, and less than 5% incidence was recorded in ICG5323, ICG2866, NRCG 1015, R13, and NRCG4400. In Jagtial, Andhra Pradesh State, 242 groundnut genotypes were evaluated during 1996–1997 for resistance to GBNV, and 10 genotypes were identified as promising resistant sources (ICGV 92269, 89/94-3-2, ICGV 91229, ICGV 91193, 89/94-7-3, 83/151-7, 85/203-6, ICGV 91248, ICGV 91117, and ICGV 86031) (25). Field screening of 172 genotypes at the Regional Research Station, Raichur, Karnataka during 1996–1997 to 1998–1999 resulted in identification of seven genotypes viz. DRG-18, ICG-7812, ICG (FDRS)-10, ICGV-80325, JSSP-3, KGN-22, and PI-393516 with high level of resistance (0 to 1% disease incidence) (28). Forty-four groundnut lines were evaluated for their resistance to GBNV in a field experiment conducted in Jalgaon, Maharashtra, during the 2000 summer season, and six lines were found highly resistant (103). Several groundnut cultivars and breeding lines (ICGV 86029, 86031, 86388, 91239, 91245, 91246, and 91249) were identified at International Crops Research Institute for the Semi-Arid-Tropics with field resistance to GBNV, and GBNV-resistant groundnut cultivars ICGS 44 and ICGS 11 were released in India. Pratap Mungphali 2, a new Spanish groundnut cultivar, was bred and developed from the cross ICGV 86055 × ICG (FDRS)-10, which was moderately resistant to GBNV in Rajasthan (59). TCGS-635, a pentafoliate groundnut cultivar derived from the F3 generation of a cross between Tirupati-1 and ICGV-86398, was found to have tolerance to GBNV at Tirupati, Andhra Pradesh (110).

Several wild *Arachis* germplasm were evaluated for resistance to GBNV, and one accession each of *A. benensis* and *A. cardenasii,* two accessions of *A. villosa* (section Arachis), two accessions of *A. appressipila* (section Procumbentes), and one accession of *A. triseminata* (section Triseminatae) were identified as resistant to GBNV. These seven field-resistant accessions, when tested under glasshouse conditions by mechanical sap inoculation, exhibited only local symptoms, and the virus was not detected in the newly developed leaves. Therefore, resistance in these accessions appears to be due to inhibition in systemic movement of the virus (75).

Resistance in mungbean. Of 38 genotypes screened for resistance to *T. palmi* and GBNV under field conditions in Hyderabad, Andhra Pradesh during 2000 winter and 2001 summer season, LGG 460, 480, 491, and 582 consistently showed resistance to *T. palmi* and GBNV (99).

Resistance in soybean. Of 48 genotypes screened against bud blight caused by GBNV during 2000 summer, genotypes MACS-754, NRC-55, VLS-55, JS-SH-96-04, TS-128-5, DSb-228, and SL-528 were highly resistant with 0.1 to 1% final disease incidence (51).

Resistance in tomato. Sources of resistance to TSWV in different *Lycopersicon* species have been identified. Genotypes with the *Sw-5* gene are more durable and the most widely used (12,14,85,102). Tomato genotypes with *Sw-5* genes when evaluated for resistance to GBNV both under natural and artificial mechanical inoculation conditions failed to show resistance against GBNV in India (M. Krishnareddy, *unpublished results*). Venkata Ramamana et al. (112) evaluated 20 cultivars, 36 genotypes, and 7 wild species of tomato for resistance to GBNV under natural and glasshouse conditions, of which EC8630 and EC5888 showed resistant reactions.

Resistance in potato. Of 207 exotic accessions of cultivated potato screened under natural conditions in Gwalior (Madhya Pradesh) for three successive years for GBNV, several accessions showed resistant reaction and can be of use in potato breeding programs for developing GBNV-resistant varieties (97).

Concluding Remarks and Research Needs

Of five tospoviruses known to occur in India, GBNV and WBNV are widely prevalent and becoming increasingly important in vegetables. TSWV and *Impatiens necrotic spot virus*, two of the well-established tospoviruses on vegetables and ornamentals (63), are not known to occur in India. Their potential introduction through ornamentals needs to be monitored. It is possible that many more tospovirus species, which are apparently not distinguishable by symptoms alone, will be identified in the subcontinent in the near future, largely due to the agricultural trade. In other countries in the subcontinent, there is a need for systematic and regular surveys to identify and document thrips vectors and tospoviruses and their impact.

The natural host range of some tospoviruses is increasing (63). WBNV, predominantly considered to be a pathogen of cucurbitaceous hosts, was recently detected in chili and tomato (47). Similarly, the possibility of natural infection of GBNV on cucurbita-

ceous hosts can't be ruled out. The host range of recently recorded CaCV and IYSV need to be further examined. Expansion in host range has resulted in mixed infection of GBNV, WBNV, and CaCV in tomato and chili (47). Their detection in mixed infection is difficult using polyclonal antibodies. There is a need to develop virus species-specific monoclonal antibodies or multiplex RT-PCR based diagnosis. Disease diagnosis becomes even more difficult when there is co-infection of GBNV with *Tobacco streak virus* (76,111).

Although GBNV and WBNV populations originating from different hosts and locations are homogenous, virus–vector or virus–host interactions have not been extensively studied in the region. Limited or no information is available about biologically distinct strains of GBNV or tospoviruses reported from India. *T. palmi* tends to be the predominant thrips vector species, but data on population dynamics and biotype composition are lacking. This information will be highly useful in refining chemical sprays for thrips control and possibly provide important parameters for developing forecasting models. Multiple and overlapping crops that are hosts to both thrips vectors and tospoviruses and nearly year-round cultivation of crops without any crop-free period provide the continuous green bridge for both the virus and vector complex, thereby making it extremely difficult if not impossible to break the 'life cycle' of the virus. Therefore, IPM tactics should focus on keeping disease incidence to the minimum so that maximum yield could be attained with minimal crop loss.

Management of the tospovirus diseases through application of cultural practices and chemical measures for thrips control is to some extent effective, but development and deployment of resistant cultivars should be the priority and should be the main tactic in developing IPM programs. In the absence of suitable resistant cultivars, an integrated disease management approach is the best option for the management of tospovirus diseases (36). Chemical control of thrips, phytosanitary cultural practices, and use of resistant cultivars have resulted in reduced disease incidence of GBNV in groundnut and WBNV in watermelon. Virus-, region-, and crop-specific IPM programs need to be developed, as one set of IPM tactics effective for one crop may not be equally effective for other crops.

Breeding for resistance, which has largely relied on the natural occurrence of the virus under field conditions, has occasionally led to misidentification of resistant sources. Greenhouse-based screening through mechanical or thrips-mediate transmission is desirable. However, GBNV and WBNV are not easily sap transmitted to tomato and watermelon, and as such optimization in transmission technique is required to develop reliable screening protocols. In addition to the conventional breeding, pathogen-derived resistance against GBNV through genetic transformation using the N gene has been attempted in some important crops including tomato and groundnut in association with private industry (65,72). These efforts have yielded transgenic lines, characterized under controlled greenhouse conditions, that are awaiting evaluation under field conditions. Virus resistant transgenic crops hold great promise for reducing the impact of tospoviruses in the Indian subcontinent.

Acknowledgments

We thank Mr. Basavaraj and Mr. Somnath for their help in preparation of this manuscript and Dr. D. V. R. Reddy and Dr. Prem Rajagopalan for critical comments on the manuscript.

Literature Cited

1. Akanda, A. M. 1994. Virus disease of tomato and eggplant and their control. Lecture note from the Trainers Training programs on grafting technology of tomato and eggplant. IPSA-JICA publication. IPSA, Salna, Gazipur, Bangladesh. pp. 28-33.
2. Akram, M., Jain, R. K., Chaudhary, V., Ahlawat, Y. S., and Khurana, S. M. P. 2004. Comparison of *Groundnut bud necrosis virus* isolates based on movement protein (NSm) gene sequences. Ann. Appl. Biol. 145:285-289.
3. Akram, M., and Naimuddin. 2009. Movement protein gene sequence based characterization of Groundnut bud necrosis virus infecting cowpea. J. Food Legumes 22:82-85.
4. Akram, M., and Naimuddin. 2010. First report of *Groundnut bud necrosis virus* infecting pea (*Pisum sativum*) in India. New Dis. Rep. 21:10.
5. Amin, P. W. 1985. Apparent resistance of groundnut cultivar Robut 33-1 to bud necrosis disease. Plant Dis. 69:718-719.
6. Amin, P. W., Ghanekar, A. M., and Reddy, D. V. R. 1979. Leaf curl disease of mung and urd bean caused by *Tomato spotted wilt virus*. Indian Phytopathol. 32:163.
7. Ananthakrishnan, T. N., and Annadurai, R. S. 2007. Thrips–tospovirus interactions: Biological and molecular implications. Curr. Sci. 92:1083-1086.
8. Anjaneya Reddy, B., Krishnareddy, M., Jalali, S., Patil, M. S., and Usha Rani, T. R. 2008. Detection of a tospovirus-infecting tomato (*Solanum lycopersicon* L.). Indian J. Virol. 19:1-5.
9. Anonymous. 2008. Annual Report (2008-09) of All India Coordinated Research Project Vegetable crops. Indian Institute of Vegetable Research, Varanasi, U. P., India. pp. 139-142.
10. Ariyarathne, I., Liyanage, T., and Sagarika, M. 2004. Virus disease incidence, alternate hosts of pathogens and disease resistance in tomato in Sri Lanka. Ann. Sri Lankan Dep. Agric. 6:13-28.
11. Bhat, A. I., Jain, R. K., Varma, A., and Lal, S. K. 2002. Nucleocapsid protein gene sequence studies suggest that soybean bud blight is caused by a strain of *Groundnut bud necrosis virus*. Curr. Sci. 82:1389-1392.
12. Boiteux, L. S., and Giordano, L. 1993. Genetic basis of resistance against two tospovirus species in tomato (*Lycopersicum eculentum*). Euphytica 71:151-154.
13. Chitturi, A. 2010. *Thrips tabaci* (Thysanoptera: Thripidae) and *Iris yellow spot virus* (genus: *Tospovirus*, family: *Bunyaviridae*) in onions: Biology and transmission studies. Ph.D. thesis. The University of Georgia, Athens. p. 201.
14. Cho, J. J., Custer, D. M., Brommonschenkel, S. H., and Tanksley, S. D. 1996. Conventional breeding: Host-plant resistance and the use of molecular markers to develop resistance to *Tomato spotted wilt virus* in vegetables. Acta. Hortic. 73:367-378.
15. Delfosse, P., Bashir, M., Malik, S. N., and Reddy, A. S. 1995. Survey of groundnut virus diseases in Pakistan. Int. Arachis Newsl. 15:51-52.
16. Desai, S. A. 1998. Reaction of peanut genotypes to bud necrosis tospovirus in Karnataka. Karnataka J. Agril Sci. 11:831-833.
17. Farooq, A., and Akanda, A. M. 2007. Symptoms and prevalence of *tomato spotted wilt virus* (TSWV) infection in Bangladesh. Int. J. Sustain. Crop Prod. 2:51-58.
18. Fauquet, C. M., Mayo, M. A., Maniloff, J., Desselberger, U., and Ball, L. A. 2005. Virus taxonomy. VIIIth Report of ICTV. Academic Press, New York. p.1259.
19. Funderburk, J. 2002. Ecology of thrips. Pages 121-128 in: Thrips and Tospoviruses. Proc. 7th Int. Sympos. Thysanoptera. R. Marullo and L. A. Mound, eds. Australian National Insect Collection, Canberra.
20. Gawande, S. J., Khar, A., and Lawande, K. E. 2010. First report of *Iris yellow spot virus* on garlic in India. Plant Dis. 94:1066.
21. Gent, D. H., du Toit, L. J., Fichtner, S. F., Krishna Mohan, S., Pappu, H. R., and Schwartz, H. F. 2006. Iris yellow spot virus: An emerging threat to onion bulb and seed production. Plant Dis. 90:1468-1480.
22. Gera, A., Kritzman, A., Cohen, J., and Raccah, B. 1998. Tospovirus infecting bulb crops in Israel. Pages 86-87 in: Recent Progress in Tospovirus and Thrips Research. D. Peters and R. Goldbach, eds. 4th Int. Sympos. Tospoviruses Thrips Floral Vegetable Crops. Wagengingen, The Netherlands.
23. Ghanekar, A. M., Reddy, D. V. R., Lizuka, N., Amin, P. W., and Gibbons, R. W. 1979. Bud necrosis of groundnut (*Arachis hypogaea*) in India caused by *Tomato spotted wilt virus*. Ann. Appl. Biol. 93:173-179.
24. Goldbach, R., and Kuo, G. 1996. Introduction. Proc. Int. Sympos. Tospoviruses Thrips Floral Vegetable Crops. Taiwan Agricultural Research Institute, Taichung, Taiwan. Acta. Hortic. 431:21-26.
25. Gopal, K., Jagadeswar, R., Babu, G. P., and Upadhyaya, H. D. 2004. Sources of resistance to bud necrosis disease in groundnut. Int. Arachis Newsl. 24:36-38.
26. Gopal, K., Krishnareddy, M., Reddy, D. V. R., and Muniappa, V. 2010. Transmission of *Peanut yellow spot virus* (PYSV) by thrips, *Scirtothrips dorsalis* Hood in groundnut. Arch. Phytopathol. Plant Prot. 43:421-429.
27. Gowda, S., Satyanarayana, T., Naidu, R. A., Mushegian, A., Dawson, W. O., and Reddy, D. V. R. 1998. Characterization of the large (L) RNA of peanut bud necrosis tospovirus. Arch. Virol. 143:2381-2390.
28. Gururaj, S., Kenchanagoudar, P. V., and Naragund, V. B. 2002. Identification of sources for field resistance to peanut bud necrosis disease in groundnut. Karnataka J. Agril. Sci.15:646-648.
29. Gururaj, S., Kenchanagoudar, P. V., Naragund, V. B., and Naik, M. K. 2005. Management of peanut bud necrosis disease through intercropping. Indian Phytopathol. 58:207-211.
30. Hemalatha, V., Pradnya, G., Anjali, A. K., Krishnareddy, M., and Savithri, H. S. 2008. Monoclonal antibodies to the recombinant nucleocapsid protein of a *Groundnut bud necrosis virus* infecting tomato in Karnataka and their use in profiling the epitopes of Indian tospovirus isolates. Curr. Sci. 95:952-957.
31. Jain, R. K., Bag, S., Umamaheswaran, K., and Mandal, B. 2007. Natural infection by Tospovirus of cucurbitaceous and fabaceous vegetable crops.

J. Phytopathol. 155:22-25.
32. Jain, R. K., Khurana, S. M. P., Bhat, A. I., and Chaudhary, V. 2004. Nucleocapsid protein gene sequence studies confirm that potato stem necrosis is caused by a strain of *Groundnut bud necrosis virus*. Indian Phytopathol. 57:169-173.
33. Jain, R. K., Pandey, N. A., Krishnareddy, M., and Mandal, B. 2005. Immunodiagnosis of groundnut and watermelon bud necrosis viruses using polyclonal antiserum to recombinant nucleocapsid protein of *Groundnut bud necrosis virus*. J. Virol. Methods 130:162-164.
34. Jain, R. K., Pappu, H. R., Pappu, S. S., Krishanareddy, M., and Vani, A. 1998. Watermelon bud necrosis tospovirus is a distinct virus species belonging to serogroup IV. Arch. Virol. 143:1637-1644.
35. Jain, R. K., Umamaheswaran, K., Bhat, A. I., Thien, H. X., and Ahlawat, Y. S. 2002. Necrosis disease on cowpea, mungbean and tomato is caused by *Groundnut bud necrosis virus*. Indian Phytopathol. 55:354.
36. Jones, D. R. 2005. Plant viruses transmitted by thrips. Eur. J. Plant Pathol. 113:119-157.
37. Jones, R. A. C., and Sharman, M. 2005. *Capsicum chlorosis virus* infecting *Capsicum annuum* in the East Kimberley region of Western Australia. Aust. Plant Pathol. 34:397-399.
38. Kato, K., Hanada, K., and Kameya-Iwaki, M. 2000. *Melon yellow spot virus*: A distinct species of the genus Tospovirus isolated from melon. Phytopathology 90:422-426.
39. Khurana, S. M. P., Phadtare, S. G., Garg, I. D., Singh, M. N., and Bharadwaj, V. P. 1989. Potato stem necrosis epidemic due to *Tomato spotted wilt virus* in India. (Abstr. p. 301) Proc. IV Int. Plant Virus Epidemiol. Workshop, Montpellier, France.
40. Krishnamoorthy, A., Ganga-Visalakshi, P. N., Manoj-Kumar, A., and Mani, M. 2007. Influence of some pesticides on entomopathogenic fungus *Lecanicillium lecanii*. J. Hortic. Sci. 2:53-57.
41. Krishnamoorthy, A., Ganga-Visalakshi, P. N., and Pillai, K. G. 2009. *Orius tantillus* against *Scirtothrips dorsalis* on capsicum under polyhouse conditions in India. In: III Int. Sympos. Biol. Control Arthropods. Lincoln University, Christchurch, New Zealand.
42. Krishnareddy, M., Jalali, S., and Venugopalan, R. 2007. Diagnosis, epidemiology and management of *Watermelon bud necrosis virus* infecting watermelon. Page 66 in: 10th Int. Plant Virus Epidemiol. Sympos. Controlling Epidemics Emerging Established Plant Virus Dis.-The Way Forward. ICRISAT, Hyderabad, India.
43. Krishnareddy, M., Usha-Rani, R., Kumar, A., Madhavireddy, K., and Pappu, H. R. 2008. First report of *Capsicum chlorosis virus* (Genus *Tospovirus*) infecting chili pepper (*Capsicum annuum)* in India. Plant Dis. 92:1469.
44. Krishnareddy, M., and Varma, A. 1990. Prevalence of *Tomato spotted wilt virus* in Delhi. Indian Phytopathl. 43:252.
45. Krishna Kumar, N. K., Venkatesh, N., Kalleshwaraswamy, C. M., and Ranganalh, H. R. 2006. Seasonal incidence of thrips and bud necrosis virus on watermelon. Pestmanag. Hortic. Ecosyst. 12:85-92.
46. Kumar, R., Mandal, B., Geetanjali, A. S., Jain, R. K., and Jaiwal, P. K. 2010. Genome organisation and sequence comparison suggest intraspecies incongruence in M RNA of *Watermelon bud necrosis virus*. Arch. Virol. 155:1361-1365.
47. Kunkalikar, S. R., Sudarsana, P., Arun, B. M., Rajagopalan, P., Chen, T.-C., Yeh, S.-D., Naidu, R. A., Zehr, U. B., and. Ravi, K. S. 2011. Importance and genetic diversity of vegetable-infecting tospoviruses in India. Phytopathology 101:367-376.
48. Kunkalikar, S. R., Sudarsana, P., Rajagopalan, P., Zehr, U. B., Naidu, R. A., and Ravi, K. S. 2007. First report of *Capsicum chlorosis virus* in tomato in India. Plant Health Prog. doi:10.1094/PHP-2007-1204-01-BR.
49. Kunkalikar, S. R., Sudarsana, P., Rajagopalan, P., Zehr, U. B., and Ravi, K. S. 2010. Biological and molecular characterization of *Capsicum chlorosis virus* infecting chili and tomato in India. Arch. Virol. 155:1047-1057.
50. Lakshmi, K. V., Wightman, J. A., Reddy, D. V. R., Ranga-Rao, G. V., Buiel, A. A. M., and Reddy, D. V. R. 1995. Transmission of *Peanut bud necrosis virus* by *Thrips palmi* in India. Pages 179-184 in: Thrips Biology and Management. B. L. Parker, M. Skinner, and T. Lewis, eds. Plenum Press, New York.
51. Lal, S. K., Bhat, A. I., Rana, V. K. S., Sara, R. I., and Kumar, A. 2002. Identification of resistance sources against bud blight disease of soybean. Indian J. Genet. Plant Breed. 62:357-358.
52. Li, T. J., Yeh, C. Y., Yeh, D. S., Joseph, A., Raja, J., Rajgopalan, A. P., Liu, Y. L., and Chen, C. T. 2010. Complete genome sequence of *Watermelon bud necrosis virus*. Arch. Virol.156:359-362.
53. Lin, Y. H., Chen, T. C., Hsu, H. T., Liu, F. L., Chu, F. H., Chen, C. C., Lin, Y. Z., and Yeh, S. D. 2005. Serological comparison and molecular characterization for verification of Calla lily chlorotic spot virus as a new tospovirus species belong to *Watermelon silver mottle virus* serogroup. Phytopathology 95:1482-1488.
54. Lokesh, B., Rashmi, P. R., Amruta, B. S., Srisathiyanarayanan, D., Murthy, M. R. N., and Savithri, H. S. 2010. NSs encoded by *Groundnut bud necrosis virus* is a bifunctional enzyme. PLoS ONE 5(3):e9757. doi:10.1371/journal. pone.0009757.
55. Mandal, B., and Jain, R. K. 2010. ELISA kit for tospoviruses detection. ICAR News 16:13.
56. Mandal, B., Jain, R. K., Chaudhary, V., and Varma, A. 2003. First report of natural infection of *Luffa acutangula* by *Watermelon bud necrosis virus* in India. Plant Dis. 87:598.
57. Meena, R. L., Ramasubramanian, T., Venkatesan, S., and Mohankumar, S. 2005. Molecular characterization of Tospovirus transmitting thrips population from India. Am. J. Biochem. Biotechnol. 1:168-173.
58. Nagata, T., Inoue-Nagata, A. K., van Lent, J., Goldbach, R., and Peters, D. 2002. Factors determining vector competence and specificity for transmission of *Tomato spotted wilt virus*. J. Gen. Virol. 83:663-671.
59. Nagda, A. K., and Dashora, A. 2005. New groundnut variety Pratap Mungphali-2 released in Rajasthan, India. Int. Arachis Newsl. 25:15-17.
60. Naidu, R. A. 2011. Management of *Peanut bud necrosis virus* disease in tomato in South Asia. (Abstr.) Phytopathology 101:S210.
61. Narayanasamy, P., Kandaswamy, T. K., and Ramiah, M. 1975. A new disease of groundnut in Tamil Nadu. Madras Agric. J. 62:371-375.
62. Nariani, T. K., and Dhingra, K. L. 1963. A mosaic disease of groundnut (*Arachis hypogaea* L.). Indian J. Agril. Sci. 33:25-27.
63. Pappu, H. R., Jones, R. A. C., and Jain, R. K. 2009. Global status of tospovirus epidemics in diverse cropping systems: Successes achieved and challenges ahead. Virus Res. 141:219-236.
64. Persley, D. M., Thomas, J. E., and Sharman, M. 2006. Tospoviruses–An Australian perspective. Aust. Plant Pathol. 35:161-180.
65. Phaneendra, Ch., Radhakrishnan, T., and Jain, R. K. 2010. Nucleocapsid and coat protein mediated strategy to confer resistance against stem and bud necrosis diseases of groundnut. Indian J. Virol. (Suppl. 1):A72.
66. Prasada-Rao, R. D. V. J., Chakraborthy, S. K., Rajeswari, R., and Reddy, A. S. 1987. Occurrence of *Tomato spotted wilt virus* on cow pea (*Vigna unguiculata* (L.) WALP.), chili (*Capsicum annum* L.) and brinjal (*Solanum melongena* L.). Indian J. Plant Prot. 15:117-119.
67. Prasada Rao, R. D. V. J., Izuka, M., Ragunathan, V., and Joshi, N. C. 1980. Incidence of *Tomato spotted wilt virus* on tomato in Andhra Pradesh. Indian Phytopathol. 33:436-439.
68. Prasada-Rao, R. D. V. J., Rajeswari, R., Rao, M. V. B., Ragunathan, V., and Joshi, N. C. 1985. Spotted wilt of pea in India. Indian Phytopathol. 38:90-93.
69. Premachandra, W. T. S. D., Borgemeister, C., Maiss, E., Knierim, D., and Poehling, H. M. 2005. Ceratothripoides claratris, a new vector of a *Capsicum chlorosis virus* isolate infecting tomatoes in Thailand. Phytopathology 95:659-663.
70. Raja, P. 2005. Development of nucleocapsid mediated resistance in tomato against *Groundnut bud necrosis virus*. Ph.D. thesis. Indian Agricultural Research Institute, New Delhi.
71. Raja, P., and Jain, R. K. 2006. Molecular diagnosis of *Groundnut bud necrosis virus* causing bud blight of tomato. Indian Phytopathol. 59:359-362.
72. Raja, P., and Jain, R. K. 2008. Development of nucleocapsid gene mediated resistance in tomato against *Groundnut bud necrosis virus*. J. Plant Pathol. 90:S2.442.
73. Ramapandu, S., and Raychaudhuri, S. P. 1980. Reaction of groundnut varieties to bud necrosis disease. Madras Agric. J. 67:559-560.
74. Ravi, K. S., Kitkaru, A. S., and Winter, S. 2006. *Iris yellow spot virus* in onions: A new tospovirus record form India. Plant Pathol. 55:288.
75. Reddy, A. S., Reddy, L. J., Mallikarjuna, N., Abdurahman, M. D., Reddy, Y. V., Bramel, P. J., and Reddy, D. V. R. 2000. Identification of resistance to *Peanut bud necrosis virus* (PBNV) in wild *Arachis* germplasm. Ann. Appl. Biol. 137:135-139.
76. Reddy, A. S., Prasada Rao, R. D. V. J., Thirumala Devi, K., Reddy, S. V., Maya, M. A., Roberts, I., Satyanarayana, T., Subramaniam, K., and Reddy, D. V. R. 2002. Occurrence of *Tobacco streak virus* in peanut (*Arachis hypogaea*) in India. Plant Dis. 86:173-178.
77. Reddy, D. V. R. 1998. Control measures for the economically important peanut viruses. Pages 541-546 in: Plant Virus Disease Control. A. Hadidi, R. K. Khetarpal, and A. Koganezawa, eds. American Phytopathological Society, St. Paul, MN.
78. Reddy, D. V. R., Amin, P. W., McDonald, D., and Ghanekar, A. M. 1983. Epidemiology and control of groundnut bud necrosis and other diseases of legumes crops in India caused by tomato spotted wilt virus. Pages 93-102 in: Plant Virus Epidemiology. R. T. Plumb and J. M. Thresh, eds. Blackwell Scientific Publications Oxford, UK.
79. Reddy, D. V. R., Buiel, A. A. M., Satyanarayana, T., Dwivedi, S. L., Reddy, A. S., Ratna, A. S., Vijaylakshmi, K., Ranga Rao, G. V., Naidu, R. A., and Wightman, J. A. 1995. Peanut budnecrosis virus disease: An overview. Pages 3-7 in : Recent Studies on Peanut Bud Necrosis Disease: Proceedings of a Meeting, 20 March. A. A. M. Buiel, J. E. Parevliet, and J. M. Lenne, eds. International Crop Research Institute for Semi-Arid Tropics, Patancheru, Andhra Pradesh, India.
80. Reddy, D. V. R., Ratna, A. S., Sudarshana, M. R., and Kiran Kumar, I. 1992. Serological relationships and purification of bud necrosis virus, a tospovirus occurring in peanut (*Arachis hypogaea L.*) in India. Ann. Appl. Biol. 120:279-286.
81. Reddy, D. V. R., Sudarshana, M. R., Ratna, A. S., Reddy, A. S., Amin, P. W., Kumar, I. K., and Murthy, A. K. 1991. The occurrence of yellow spot virus, a member of tomato spotted wilt virus group, on peanut (*Arachis hypogaea* L) in India. Pages 77-88 in: Virus-Thrips-Plant Interactions of to-

mato spotted wilt virus. H. T. Hsu and R. H. Lawson, eds. Proc. USDA Agricultural Research Service, ARS-87.
82. Reddy, D. V. R., Wightman, J. A., Beshear, R. J., Highland, B., Black, M., Sreenivasulu, M., Dwivedi, S. L., Demski, J. W., McDonald, D., Smith, J. W., Jr., and Smith, D. H. 1991. Bud necrosis: A disease of groundnut caused by *Tomato spotted wilt virus*. Page 20 in: Inf. Bull. No. 31. International Crop Research Institute for the Semi Arid Tropics, Patancheru, Andhra Pradesh, India.
83. Reddy, M., Reddy, D. V. R., and Appa Rao, A. 1968. A new record of virus disease on peanut. Plant Dis. Rep. 52:494-495.
84. Riley, D., Shimat, J., Srinivasan, R., and Diffie, S. 2011. Thrips vectors of tospoviruses. J. Integ. Pest Manag. 1(2): DOI: 10.1603/IPM10020.
85. Rosell'o, S., Diez, M. J., and Nuez, F. 1996. Viral diseases causing the greatest economic losses to tomato crop. I. The *Tomato spotted wilt virus*: A review. Sci. Hortic. 67:117-150.
86. Saritha, R. K. 2007. Biological characterisation and genome organization of *Groundnut bud necrosis virus* from mungbean (*Vigna radiata*). Ph.D. thesis. Indian Agricultural Research Institute, New Delhi.
87. Saritha, R. K., and Jain, R. K. 2007. Nucleotide sequence of the S and M RNA segments of a *Groundnut bud necrosis virus* isolate from *Vigna radiata* in India. Arch. Virol. 152:1195-1200.
88. Satyanaryana, T., Gowda, S., Lakshminaryan Reddy, K., Metals, E., Dawson, W. O., and Reddy, D. V. R. 1998. *Peanut yellow spot virus* is a member of a new serogroup of Tospovirus genus based on small (S) RNA sequence and organization. Arch. Virol. 143:353-364.
89. Satyanaryana, T., Lakshminaryan Reddy, K., Ratna, A. S., Deom, C. M., Gowda, S., and Reddy, D. V. R. 1996. *Peanut yellow spot virus*: A distinct tospovirus species based on serology and nucleic acid hybridization. Ann. Appl. Biol. 129:237-245.
90. Satyanaryana, T., Mitchell, S. E., Reddy, D. V. R., Brown, S., Kresovich, S., Jarret, R., Naidu, R. A., and Damski, J. W. 1996. Peanut bud necrosis tospovirus S RNA: Complete nucleotide sequence, genome organization and homology to other tospoviruses. Arch. Virol. 141:85-98.
91. Satyanaryana, T., Mitchell, S. E., Reddy, D. V. R., Brown, S., Kresovich, S., Jarret, R., Naidu, R. A., and Damski, J. W. 1996. The complete nucleotide sequence and genome organization of the M RNA segment of Peanut bud necrosis tospovirus and comparison with other tospoviruses. J. Gen. Virol. 77:2347-2352.
92. Sharma, B. P. 1996. Current status of research on legume viruses with special reference to groundnut viruses in Nepal. Page 32 in: Groundnut Virus Diseases in the Asia-Pacific Region. Summary and Recommendations of the 4th Meeting of the International Working Group. D. V. R. Reddy and C. L. L. Gowda, eds. ICRISAT, India.
93. Sharma, D. C. 1966. Studies on bunchy top, chlorosis and ring mottle virus diseases of groundnut (*Arachis hypogaea* L.). Phytopathol. Z. 57:127-137.
94. Singh, A. B., and Srivatava, S. K. 1995. Status and control strategy of peanut bud necrosis disease in Uttar Pradesh. Pages 65-68 in: Recent Studies on Peanut Bud Necrosis Disease: Proceedings of a meeting, 20 March. A. A. M. Buiel, J. E. Parevliet, and J. M. Lenne. eds. International Crop Research Institute for Semi-Arid Tropics, Patancheru, Andhra Pradesh, India.
95. Singh, R. B., Srivastava, K. K., Khurana, S. M. P., and Pandey, S. K. 1997. Assessment of yield losses due to potato stem necrosis disease. Indian J. Virol. 3:135-137.
96. Singh, S. J., and Krishnareddy, M. 1996. Watermelon bud necrosis: A new Tospovirus disease. Acta Hortic. 431:68-77.
97. Somani, A. K., and Gopal, J. 2004. Screening of exotic potato germplasm for resistance to stem necrosis. Indian Phytopathol. 57:61-64.
98. Somani, A. K., Kushwa, V. S., Khurana, S. M. P., and Garg, I. D. 2007. Integrated management of stem necrosis disease of potato in Central India. Potato J. 34:231-234.
99. Sreekanth, M., Sreeramulu, M., Rao, R. D. V. J., Babu, B. S., and Babu, T. R. 2002. Evaluation of green gram genotypes for resistance to *Thrips palmi* and *Peanut bud necrosis virus*. Indian J. Plant Prot. 30:109-114.
100. Sreekanth, M., Sreeramulu, M., Rao, R. D. V. J., Babu, B. S., and Babu, T. R. 2004. Effect of intercropping on *Thrips palmi* population and *Peanut bud necrosis virus* incidence in mungbean. Indian J. Plant Prot. 32:45-48.
101. Sreekanth, M., Sriramulu, M., Rao, R. D. V. J. P., Babu, B. S., and Babu, T. R. 2004. Evaluation of certain new insecticides against *Thrips palmi* (Karny), the vector of *Peanut bud necrosis virus* (PBNV) on mungbean (*Vigna radiata* L. *Wilczek*). Int. Pest Cont. 46:315-317.
102. Stevens, M. R., Scott, S. J., and Gergerich, R. C. 1992. Inheritance of a gene for resistance to *Tomato spotted wilt virus* (TSWV) from *Lycopersicon peruvianum* Mill. Euphytica 59:9-17.
103. Thakare, C. S., Shambharkar, D. A., Suryawanshi, R. T., and Patil, R. B. 2002. Screening of groundnut germplasm lines against peanut bud necrosis disease. J. Maharashtra Agril. Univ. 27:320.
104. Thien-Xuan, H., Bhat, A. I., and Jain, R. K. 2003. Mungbean necrosis disease is caused by a strain of Groundnut bud necrosis tospovirus. Indian Phytopathol. 56:54-60.
105. Thira, S. K., Cheema, S. S., and Kang, S. S. 2004. Pattern of bud necrosis disease development in groundnut crop in relation to different dates of sowing. Plant Dis Res. Ludhiana. 19:125-129.
106. Tsompana, M., and Moyer, J. W. 2008. Tospoviruses. Pages 157-162 in: Encyclopedia of Virology, vol. 5, 3rd ed. B. W. J. Mahy and M. H. V. Van Regenmortel, eds. Elsevier Ltd., Oxford, UK.
107. Ullman, D. E., Sherwood, J. L., and German, T. L. 1997. Thrips as vectors of plant pathogens. Pages 539-565 in: Thrips as Crop Pests. T. Lewis, ed. CAB International, Wallingford, UK.
108. Umamaheswaran, K., Jain, R. K., Bhat, A. I., and Ahlawat, Y. S. 2003. Biological and molecular charecterisation of a tospovirus isolates from tomato and its relationship with other tospoviruses. Indian Phytopathol. 56:168-173.
109. Varma, A., Jain, R. K., and Bhat, A. I. 2002. Virus resistant transgenic plants for environmentally safe management of viral diseases. Indian J. Biotechnol. 1:73-86.
110. Vasanthi, R. P. 2003. A pentafoliate groundnut (*Arachis hypogaea* L.) culture, TCGS-635. Indian J. Genet. Plant-Breed. 63:169-170.
111. Vemana, K., and Jain, R. K. 2010. New experimental hosts of *Tobacco streak virus* and absence of true seed transmission in leguminous hosts. Indian J. Virol. 21:117-127.
112. Venkata Ramamana, C., Rao, R. D. V. J., Reddy, I. P., Venkata Rao, P., and Reddy, Y. N. 2006. Screening of tomato germplasm and wild relatives against *Peanut bud necrosis virus* (PBNV) Disease. Indian J. Plant Prot. 34:59-61.
113. Whitfield, A. E., Ullman, D. E., and German, T. L. 2005. Tospovirus-thrips interactions. Annu. Rev. Phytopathol. 43:459-489.
114. Wightman, J. A., and Amin, P. W. 1988. Groundnut pests and their control in the semi-arid tropics. Trop. Pest Manag. 34:218-226.

Bikash Mandal

R. K. Jain

M. Krishnareddy

N. K. Krishna Kumar

K. S. Ravi

Hanu R. Pappu

Dr. Mandal is a senior scientist at the Advanced Centre for Plant Virology, Indian Agricultural Research Institute (IARI), New Delhi. Dr. Mandal obtained his B.S. (1989) from Bidhan Chandra Krishi Viswa Vidyalaya, Mohanpur, his M.S. (1990) in plant pathology (virology) from University of Agricultural Sciences, Bangalore, and his Ph.D. (1994) in plant pathology (virology) from IARI. His major research area is on vegetable viruses in India with focus on characterization, diagnosis, and transgenic resistance. He worked on tospoviruses in southern Georgia, U.S.A., and in India. He is currently a consortium principal investigator of a World Bank funded National Innovation Project (NAIP) on novel strategies for molecular diagnosis of plant viruses. Dr. Mandal teaches plant virology at IARI.

Dr. Jain is head of the Division of Plant Pathology at Indian Agricultural Research Institute (IARI), New Delhi, India. He received his Ph.D. (1988) in plant pathology from IARI, New Delhi, and subsequently postdoctoral experience from CSIRO, Melbourne, Australia (1991) and University of Georgia, Tifton, U.S.A. (1997–98). Over the last 20 years, he has worked on chronic and emerging poty-, ilar-, and tospoviruses affecting field and horticultural crops. His expertise lies in the area of diagnosis, genomics, and transgenics against viruses.

Dr. Krishnareddy received his M.Sc. (Ag) in plant pathology and his Ph.D. in plant pathology (virology) from the Indian Agricultural Research Institute, New Delhi, India. He then joined as a scientist the Indian Institute of Horticultural Research (IIHR) of Indian Council of Agricultural Research (ICAR) at Bangalore, India. His postdoctoral experience was working with molecular cloning and expression of cellulose degrading enzymes from *Clostridium thermocellus* at National Food Research Institute, Tsukuba, Japan under United Nations University fellowship. As a plant virologist, he continues to work on viruses of fruits, vegetables, and ornamental crops. His main research interests include molecular characterization and diagnosis of begomoviruses, ilarviruses, potyviruses, and tospoviruses and development of virus-resistant varieties/hybrids in vegetable crops and integrated management of viruses in horticultural crops. He currently serves as principal scientist and head of the Division of Plant Pathology, IIHR, Bangalore.

Dr. Krishna Kumar has been working at Indian Institute of Horticultural Research (ICAR), Bengaluru. He has been associated with entomology and IPM of vegetables for the last three decades. His major area of specialization is virus–vector relationships. He obtained his doctoral degree from University of Hawaii under Dr. Diane E. Ullman, a renowned entomologist, and did his postdoctoral research on thrips transmission of *Tomato spotted wilt virus* (TSWV) at UC Davis. He has organized a number of national meetings on IPM in horticulture, thrips–tospovirus interactions, and group meetings on sucking pests. He is also the founder member of the Indian Academy of Entomology. He has also handled a network project on sucking pests funded by ICAR and Co-PI for NAIP project on gene silencing in *Helicoverpa armigera*. He is an innovator in the field of molecular systematics; developed DNA barcode for 50 different species of insect pests of different orders of insects, viz. Thysanoptera, Hemiptera, Coleoptera, Lepidoptera, and Diptera; and designed species-specific markers for major insect pests of horticultural crops. He is presently working as director of the National Bureau of Agriculturally Important Insects (ICAR), Bangaluru.

Dr. Ravi is head of the Vegetable Research Center, Maharashtra Hybrid Seeds Company Ltd., Bangalore, India. He obtained his M.Sc. (Agri) and Ph.D. in plant pathology with a specialization in plant virology from the University of Agricultural Sciences (UAS), GKVK, Bangalore. He worked as a faculty member in the Departments of Plant Pathology and Biotechnology, UAS, Bangalore from 1984 to 1996. He was awarded the Rockefeller Fellowship (1996–98) to pursue a postdoctoral program in Glucoronide Inducible Gene Expression at the Center for Application of Molecular Biology to International Agriculture (CAMBIA), Canberra, Australia. In 1999, he joined Mahyco Research Center as a principal investigator and initiated a molecular virology program to work on plant viruses with special emphasis on *Begomovirus*, *Tospovirus,* and *Ilarvirus*.

Dr. Pappu is professor and chair of plant pathology and holds the President Sam Smith Distinguished Professorship in Plant Virology at Washington State University (WSU), Pullman, WA. Dr. Pappu's research interests and expertise include molecular and biotechnological approaches for characterization and control of viral diseases of horticultural crops; development of environmentally friendly and sustainable IPM strategies for reducing the impact of destructive viral diseases; capacity building in developing countries; and technology transfer. His current research focus is on biology and molecular biology of thrips-transmitted tospoviruses (genus *Tospovirus,* family *Bunyaviridae*) and biological and molecular studies of plant pararetroviruses (genus *Caulimovirus*). He is a member of the graduate faculty of the university's interdisciplinary graduate program in molecular plant sciences, and was elected secretary of the International Working Group on Viruses of Vegetables and Legumes in 2011 for a 3-year term. Dr. Pappu teaches a graduate course in plant virology and team teaches a graduate course in molecular genetics of plant-pathogen interactions and an undergraduate course on general plant pathology. As chair, he provides leadership to the statewide research, extension, and outreach programs in plant pathology and administers the graduate program (M.S. and Ph.D.) in plant pathology.