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REVIEW

Significant plant virus diseases in India and a glimpse of modern disease management technology

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Abstract India has a diverse agroclimate representing tropical, subtropical and temperate climates with zones ranging from average to high in temperature, humidity and rainfall; from low to scarce rainfall in deserts to cold to very cold plains and upland areas. Thus it is the home of one of the richest flora and fauna in the world. For pests and pathogens, too, tropical and subtropical climates are ideal for growth and development. A majority of the world's fruits, vegetables, cereals, pulses, oil seed crops, fiber crops, sugarcane, spices, and ornamentals are cultivated in India. Virus diseases and their vectors are also in abundance. Since ancient times, virus-like diseases and management practices have been known in India. This knowledge has now been revived and adopted in organic farming, especially for medicinal and aromatic plants. Current trends for extensive and intensive agriculture, open international agricultural trade, and thus food security and sustained economy have brought new challenges in the fight against virus diseases. In this changing scenario, current diseases of significance are caused by begomoviruses, badnaviruses, cucomoviruses, potyviruses, ilarviruses in crops such as vegetables, fruits, ornamentals, fiber crops, and sugarcane. Variability in the viruses is also common. Briefly reviewed here is the positive effect of an isolate of *Rice necrosis mosaic virus*; when artificially inoculated on jute and mesta fiber crops enhanced their fiber contents. Diseases of significance, e.g., leaf fleck disease of sugarcane, citrus yellow mosaic disease, banana bunchy top, banana bract mosaic, mungbean yellow mosaic, mosaic in chrysanthemum, gladiolus and orchids are also discussed. Efficient, reliable diagnostic tools have been developed and used extensively. Some of the advanced laboratories have been accredited for virus indexing under the National Certification System for tissue-cultured plants. International standards for phytosanitary measures have been promulgated, and a mandatory nodal agency is in place for the conservation and exchange of germplasm; a Containment Level-4 facility to examine incoming transgenics and a fully equipped laboratory to intercept virus-infected plant material are functional. A National Agricultural Biosecurity System will soon be in place. All these measures are essential to protect agricultural systems and to compete in the international agriculture market. Continued vigilance, disease mapping and adopting the latest technology are required to practice sustainable agriculture.

Keywords Vrikshayurveda · PCR · Multiplex PCR · ELISA · Jute · Mesta · *Badnavirus*

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Introduction

The Indian civilization is one of the oldest in world, and so is the practice of agriculture, which are several millennia old. A Sanskrit term, *vyadhi*, which is equivalent to the word disease in English, was first used in India in Krishi-Parashara compiled by sage Parashara (c. 400 BC) (Nene 2006a). Descriptions of certain specific symptoms like yellowing of leaves, closely resembling what are now known as viral diseases, have been found in Vrikshayurveda (The Science of Plant Life) written by Surapala in c. 1000 AD (Sadhale 1996). This treatise is based on the principles of Ayurveda, a millennia-old Indian system of

human health management. Ancient, medieval, and pre-modern practices used to manage these plant viral diseases have been described by Nene (2003, 2006b) and are now being practiced in several laboratories and in organic farming, especially for growing medicinal and aromatic plants.

In the modern era, the earliest recorded viral and phytoplasma diseases in India are root wilt of coconut (*Cocos nucifera* L.) present in south Kerala since 1882 (Butler 1908), spike disease of sandal (*Santalum album* L.), observed in Karnataka in 1899 (McCarthy 1903), small cardamom (*Elettaria cardamomum* Maton) mosaic (commonly known as 'Marble' or 'Katte') disease (Mollison 1900), yellow leaf disease of areca nut palm (*Areca catechu* L.) observed in 1914 (Varghese 1934), cotton stenosis (Kottur and Patel 1920), tristeza disease of citrus (Brown 1920), mosaic disease of sugarcane (Dastur 1923), and yellow vein mosaic disease of bhendi (okra; *Abelmoschus esculentus*) (Kulkarni 1924). Root wilt of coconut, spike disease of sandal, and yellow leaf of arecanut were later identified as phytoplasma diseases. In earlier review papers, I chronicled the development of modern virology in India and its important viral diseases (Rishi 2004, 2006); here I will elaborate on certain significant viral diseases of current importance and the modern technology developed for investigating viral diseases.

Bhendi yellow vein mosaic disease

Okra, popularly known as bhendi (*Abelmoschus esculentus* L.) in India is a common vegetable grown in several countries. Bhendi fruits are a rich source of proteins (20–24% edible) and minerals, and seeds contain 13–22% edible oil. Ten species of *Abelmoschus* occur in India; they are believed to be of Asiatic origin. *A. esculentus*, the cultivated species is probably of Indian origin (Dhankar et al. 2005). Bhendi, exported from India as a fresh vegetable, comprises 70% of the total fresh vegetable earnings, excluding onion (APEDA 2000). The annual yield in India is around 3.8 million tons, the highest in the world (http://www.postharvestindia.com).

Bhendi yellow vein mosaic disease (BYVMD) was first reported in 1924 (Kulkarni 1924) during the erstwhile Bombay Presidency. Different degrees of chlorosis and yellowing of veins and veinlets, smaller leaves, fewer and smaller fruits, and stunting are characteristic of BYVMD (Fig. 1a, b). Fruit yield is also greatly reduced, by as much as 96% if the crop is infected at early stage (Pun and Doraiswamy 1999). Several BYVMD-resistant bhendi varieties have been released, but none have retained resistance for long (Usha 2008).

Bhendi yellow vein mosaic disease is caused by *Bhendi yellow vein mosaic virus* (BYVMV), which is a species of genus *Begomovirus*, family *Geminiviridae* (Fauquet and Stanley 2005). BYVMV is believed to have originated in India (Usha 2008). The only known method of transmission is through whitefly (WF) (*Bemisia tabaci* Gennadius). Begomoviruses are very diverse and parallel whitefly in their geographical distribution throughout the tropics and parts of the subtropics. Begomoviruses have mono- or bipartite genomes consisting of circular single-stranded DNA of 2.6–3.0 kbp.

Bhendi is also rich in polysaccharides and polyphenols. Polysaccharides are viscous and gluelike; they inhibit *Taq* polymerase activity, thus interfering with PCR. Their texture also creates problems for DNA during pipetting. For cloning the Begomovirus genome either by directly restricting the replicative form from total nucleic acid or by amplifying the full or partial genome with Begomovirus-specific primers, high quality DNA is a prerequisite. Ikegami et al. (1981) handled this problem by extracting DNA from a nonmucilaginous host. But to characterize all the genomic components that may be involved in inciting the disease, the viral DNA should be extracted from natural host. Jose and Usha (2000) developed a protocol that is simple, cheaper and enables the isolation of pure viral DNA without ultrapurification of the virus. For extraction, they used sodium citrate buffer (pH 6.0), and for virus precipitation, PEG 6000 was added after alkali lysis. The citrate buffer eliminated the mucilage and polyphenols, and the alkali lysis enriched the replicative form of DNA. The extracted DNA could be digested with restriction enzyme and cloned successfully with no problems. PCR amplification (using begomovirus-specific primers) and cloning yielded a 2.7-kbp DNA-A of BYVMV. Despite full efforts, no second genomic component DNA-B was found, leading to the conclusion that BYVMV is monopartite. Agroinoculation with infective full-length DNA-A of BYVMV, however, only induced mild curling in bhendi.

Using non-overlapping abutting primers (beta 1.F and beta 1.R), a ~1.35-kbp DNA β fragment was amplified from BYVMV-infected bhendi plant. These abutting primers are located in the highly conserved region of DNA β sequences. When DNA β was used with DNA-A to coinoculate bhendi, typical symptoms of BYVMD resulted. This proved that BYVMD is caused by a complex of DNA-A and DNA β.

BYVMD-associated DNA β has some commonality with DNA β associated with *Ageratum yellow vein virus* and *Cotton leaf curl virus* (CLCuV) (Jose and Usha 2003). Zhou et al. (1998) concluded, on the basis of sequences of *Okra yellow vein virus* (OYVMV) and CLCuV in Pakistan, that recombination of OYVMV with another unidentified begomovirus in Okra produced a virus that caused an

epidemic in cotton. In bipartite begomoviruses, movement protein (MP) BC1 and nuclear shuttling protein (NSP) BV1 encoded by the DNA-B component induce systemic symptoms. In monopartite begomoviruses, this role is carried on either by the coat protein (CP) when it becomes highly karyophilic and active in the nuclear import/export of viral nucleoproteins across the nuclear pore complex, or a functional βC1 transcript that modulates virus-like symptoms or in some cases suppresses RNA silencing.

Kumar et al. (2006) studied protein–protein interaction and nuclear trafficking of the CP and βC1 protein associated with BYVMV. In fusion studies with green fluorescent protein (GFP), they found that GFP fused with CP (GFPCP) in the nucleus, whereas GFP fused with βC1 (GFP βC1) near the periphery of epidermal cells. Expression of βC1 in transgenic *Nicotiana benthamiana* resulted in severe abnormalities such as stunting and distorted stem and leaves. Gopal et al. (2007) showed a differential role of C4 and βC1 of BYVMV in the suppression of posttranscriptional gene silencing (PTGS) and evidence for transactivation by C2.

There are seven open reading frames (ORFs) in DNA-A and one in DNA β, that is half the size of DNA-A and functions as a symptom modulator. The highly conserved intergenic region (IR) transcribes genes in a bidirectional manner in monopartite begomoviruses. Many motifs in the IR control gene expression and replication. In a study of the functions of C2, C4 and βC1, Gopal et al. (2007) revealed that C2 is involved in transactivation and mildly suppresses gene silencing, but C4 and βC1 strongly suppress PTGS.

When inheritance of resistance to BYVMV was studied in interspecific crosses between *A. manihot* and *A. tetraphyllus*, a single dominant gene controlled resistance (Jambhale and Nerker 1981). Susceptibility to BYVMV in *A. manihot* ssp. *manihot* was controlled by two dominant genes (Sharma and Dhillon 1983). Several species (*A. manihot* [some forms], *A. pungens*, *A. crinitus*, *A. panduraeformis*, and *A. vitifolius*) were resistant to BYVMV. *A. manihot* ssp. *manihot*, *A.* spp 'Ghana', *A. tuberculatus* and *A.* spp (West African okra) were symptomless carriers of BYVMV and may be useful in developing BYVMV-resistant hybrids (Dhankar and Mishra 2004).

Mungbean yellow mosaic disease

Of all the yellow mosaic diseases of grain legumes (pulses), mungbean yellow mosaic disease (MYMD) caused by mungbean yellow mosaic virus (MYMV) is most important. MYMV induces yellow mosaic diseases in several legumes viz. *Vigna radiata* (mungbean/greengram), *V. mungo* (urdbean/blackgram), *V. aconitifolia* (mothbean), *V. unguiculata* (cowpea), *Cajanus cajan* (arhar/pigeonpea), *Glycine max* (soybean), *Phaseolus vulgaris* (French bean), and *P. lunatus* (lima bean) in India, Bangladesh, Pakistan, Sri Lanka, Thailand, Philippines, and Indonesia. Grain legumes in India are cultivated year round for both Rabi and Kharif crops because they help restore soil fertility by fixing atmospheric nitrogen. In addition, pulses are the major source of protein for the largely vegetarian population in India. The essential amino acid lysine is present in pulses in high quantities compared to the low quantities in cereals. Furthermore, the pulse protein and cereal protein are complementary, together providing a complete source of amino acids for humans.

Mungbean is native to India, where it has been cultivated and domesticated since 2200 BC. India leads Pakistan, Sri Lanka, Thailand, Laos, Kumpuchia, Vietnam, Malaysia, and China in mungbean acreage (55%) and production (45%). Due to its rapid maturity and photoinsensitivity, mungbean is adaptable to multiple cropping systems in the lowlands of the tropics and subtropics. It is easily digestible and free from flatulence-inducing factors.

MYMD is the major constraint in raising healthy, profitable crops of mungbean and other hosts of MYMV. It was first seen in New Delhi in 1955 (Nariani 1960) on mungbean, and the putative causal agent was named MYMV. It is transmitted by WF in a circulative, nonpropagative manner. In mungbean, yellow mosaic disease incidence in farmers' fields may be as high as 100%. The symptoms appear in the form of small irregular yellow specs and spots, which enlarge until leaves are completely yellowed. Diseased plants are stunted, with fewer flowers and pods that bear smaller, occasionally shriveled seeds (Fig. 1c). In severe cases, the disease may cause total loss in yield (Nariani 1960). Varma et al. (1992) reported that yellow mosaic of mungbean, urdbean, cowpea, and soybean induce an annual yield loss of US$300 million in India.

Genetics of resistance to mungbean yellow mosaic virus in mungbean was studied using four resistant cultivars (LM696, L-24-2, ML513 and ML505) and four susceptible (P9333, K891, K851 and T44). These cultivars were used as resistant and susceptible parents for a crossing program with F_1, F_2, back cross and reciprocal cross generations. The cross between resistant parents suggested that resistant parents had different resistant genes and that an inhibitory gene interaction was operating in controlling disease resistance. Crosses of susceptible parents also suggested similar genetic constitutions (Rishi 2004, 2006). Resistance was governed by two major genes with complimentary and inhibitory gene interactions. A similar study of the genetics of resistance of MYMV-cowpea isolate was done on cowpea using resistant cvs. CS39, CS55, RC80 and KBB and susceptible cvs. GC2, HFC42-1 and FS68 as parents.

In this case, resistance to the causal virus was governed by dominant gene and symbols *Cym1* and *Cym2* were assigned to the resistant genes (Rishi et al. 1996; Sangwan and Rishi 2004).

Mandal et al. (1997) cloned the DNAs of a blackgram isolate of MYMV and produced systemic infection in *V. mungo* after agroinoculation, thereby proving Koch's postulate. The progeny virus particles from the agroinoculated plants produced typical yellow mosaic symptoms when transmitted by WF on healthy blackgram seedlings. Nucleotide sequence analyses of isolates of yellow mosaic collected from different legume hosts of MYMV established the presence of *Mungbean yellow mosaic India virus* (MYMIV) on urdbean, mungbean, soybean, pigeonpea, French bean, mothbean and cowpea in northern-, central- and eastern-India, Pakistan and Bangladesh and of *Mungbean yellow mosaic virus* (MYMV) on mungbean and urdbean in western and southern India and Thailand (Girish and Usha 2005; Malathi et al. 2005; Usharani et al. 2004).

Bipartite begomoviruses have an intergenic region (IR) in the genomic component of DNA-A and DNA-B. Within the IR, a short stretch of 118–200 nucleotides, called the common region (CR), is almost identical (99–100%). Contrary to this observation in MYMV, there is no CR in the isolates of MYMIV; rather the IR of DNA-A and DNA-B is variable. MYMIV Lud-Sb isolate is the only exception with 93% identity for the CR (Periasamy 2006). After agroinoculations with the cowpea isolate (MYMIV-Cp) and blackgram isolate (MYMIV-Bg) of blackgram, cowpea, mungbean and French bean, French bean had the higher disease incidence with both the isolates, but symptoms caused by MYMIV-Bg isolate were more severe than by MYMIV-Cp. On cowpea cv. Pusa Komal, MYMIV-Bg produced mild typical leaf curl, but MYMIV-Cp caused severe leaf curl. Cowpea cv. Lal Lobia was not susceptible. MYMIV-Cp in the field caused yellow mosaic (Fig. 1d), and at advanced stages, there was complete yellowing of leaves, stunting, and fewer flowers and fruits were set. DNA β is commonly found in monopartite begomoviruses, but recently in a few cases DNA β has been reported in bipartite begomoviruses including MYMIV (Rouhibakhsh and Malathi 2005). Co-agroinoculations with MYMIV-Cp DNA-A and DNA-B components with DNA β on urdbean, cowpea, French bean and mungbean only marginally enhanced infectivity and severity of symptoms. Replicative viral forms of DNA-A and DNA-B were higher, and DNA β was extremely low (Periasamy 2006). In another study, the soybean isolate (MYMIV-Sb) was highly infectious after agroinoculations of French bean, cowpea and mungbean but had lower infectivity on urdbean and its natural host soybean. MYMIV-Sb infected cowpea after agroinoculation, but MYMIV-Bg and mungbean isolate (MYMIV-Mg) did not, indicating strain variation. MYMIV-Sb genome had 15% divergence in the CR. WF transmitted progeny virus particles of MYMIV-Sb from agroinoculated soybean plants to healthy soybean seedlings, thus confirming Koch's postulates (Usharani et al. 2004). On the basis of the complete nucleotide sequence analysis of DNA-A and DNA-B from the yellow mosaic isolates on soybean in central and southern India, the isolates were identified as *Mungbean yellow mosaic India virus*-Soybean (MYMIV-Sb) in central India and *Mungbean yellow mosaic virus*-Soybean (MYMV-Sb) in southern India. Nucleotide sequence similarity in DNA-A of these isolates was 82% and in DNA-B 71%. The analyses of the CR of these isolates indicated considerable divergence in the origin of replication (*ori*), but this did not impair their infectivity as demonstrated by agroinfection with partial tandem repeats (PTRs) of MYMIV-Sb (Girish and Usha 2005).

Movement protein (MP) and nuclear shuttling protein (NSP) are the two movement proteins encoded by the bipartite geminiviruses that facilitate the cell-to-cell movement needed for infectivity and systemic infection by plant viruses. Replication of geminiviruses takes place in the nuclei, requiring transport of the viral genome through the nuclear envelope and plasmembrane. These movement proteins interact with the viral nucleic acids in a sequence-nonspecific manner during the transport process. A recognition property of the MP (comprising 298 amino acid residues) of MYMIV-Sb was studied (Radhakrishnan et al. 2008). In *N. benthamiana* it was localized at the periphery of epidermal cells when expressed ectopically. For studying the recognition properties, the MP was expressed in *Escherichia coli* as maltose-binding fusion protein (MP-MBP), which was purified in native condition. The results of gel mobility shift assays to analyse the DNA recognition properties of purified MP-MBP indicated sequence non-specific and concentration-dependent binding of MP of MYMIV-Sb to both dsDNA and ssDNA with a high affinity with ssDNA. It did not specifically recognize plasmid DNA, but showed size selection towards linear dsDNA.

In a phylogenic study of the viral genome components involved in yellow mosaic diseases in Southeast Asia, the DNA-A phylogenic tree of MYMV formed two distinct branches; one with MYMV strains from India, Thailand and Pakistan and the other with the MYMIV strains from India, Pakistan, Bangladesh and Nepal. The phylogenic tree of DNA-B of MYMV formed three clusters, whereas that of MYMIV formed one cluster indicating homogeneity of the DNA-B population in MYMIV isolates. The phylogenic tree based on the *ori* region yielded separate clusters for the majority of DNA-A and DNA-B components because of variability in the *ori* region (Girish and

Fig. 1 Symptoms of virus-infected plants in India. **a** Initial symptoms caused by *Bhendi yellow vein mosaic virus* after natural infection of bhendi, yellowing of veins. **b** Advanced symptoms caused by *Bhendi yellow vein mosaic virus* after natural infection of bhendi, marked yellow vein network. **c** *Mungbean yellow mosaic virus* after natural infection on mungbean susceptible varieties T-44 and K-851. **d** *Mungbean yellow mosaic virus* after natural infection on cowpea susceptible varieties FS-68 and HFC-42-1. **e**, **f** Leaf fleck disease of sugarcane caused by natural infection with *Sugarcane bacilliform virus.* **g** Banana buchy top disease caused by natural infection with *Banana buchy top virus*. **h** Banana bract mosaic disease caused by natural infection with *Banana bract mosaic virus*. **i** *Citrus yellow mosaic virus* on sweet orange. **j–k** *Tomato aspermy virus* on chrysanthemum. **j** Necrotic rings and mosaic on leaves. **k** Distortion of flower, rolling and twisting of corolla. **l–m** *Cucumber mosaic virus* on gladiolus. **l** Severe mosaic on leaves. **m** Color break on flowers

Usha 2005). The only exception was the clustering of the DNA-B components from two isolates of MYMV with the DNA-A from MYMIV.

The CP is instrumental in transmitting the virus and in determining the viral DNA yield in the infected tissue. The replication initiator protein (Rep) regulates the rolling replication of viral DNA. Malik et al. (2005) studied the interaction of the CP and Rep of MYMIV and reported that CP downregulated the nicking and closing function of Rep. This result indicates the importance of CP in controlling viral DNA replication, which might be useful in disease management.

Tracking the DNA replication mechanism of plant DNA viruses in a suitable eukaryotic model system may open a new avenue for the management of DNA plant viruses. At the present time, it is difficult to manage the serious, ever-increasing problems of begomoviruses because the only known vector, WF, is developing resistance to chemical control, variability is very common in begomoviruses, large scale movement of plant materials to different areas and introduction of newer crops. Such technology is new for understanding replication of plant DNA, which has been little studied in host plants.

Raghavan et al. (2004) has reported developing a yeast model system (*Saccharomyces cerevisiae* W303a strain), which during budding supports the DNA replication of an Indian isolate of mungbean yellow mosaic begomovirus. AC5 viral factor was identified as an important contributor to viral DNA replication. This yeast model system can also be exploited for screening for inhibitors of virus replication. Rep is the most important viral protein for DNA replication. In further study with MYMIV, Chilakamarthi et al. (2007) designed an anti-Rep ribozyme to target viral DNA replication. When evaluated in *S. cerevisiae* W303a, which lacks RNAi machinery and is thus a suitable surrogate host (Raghavan et al. 2004) for replication of the virus DNA, in the presence of the anti-Rep ribozyme the yeast cell, grew slower because its growth was dependent on the begomovirus replication. Viral DNA replication also decreased by $\sim$65%. These results are encouraging for developing a virus-resistant genetically modified plant.

Rice necrosis mosaic virus induced productivity in jute and Mesta fiber crops

Rice necrosis mosaic virus (RNMV), taxonomically placed under genus *Bymovirus*, family *Potyviridae* (Shukla et al. 1994), was first seen in Japan in 1959 (Fujii 1967). In India it was first reported by Ghosh (1979). RNMV infection of rice starts as a mosaic on the emerging leaves, then the chlorotic spots enlarge into yellow chlorotic streaks and necrotic spots. Eventually, fewer plant tillers and shorter plants are produced; yields can be reduced by 12.7–100% depending upon the variety (Fujii 1967; Ghosh 1980; Inouye and Fujii 1977). On the other hand, when dicot jute crops (*Corchorus capsularis* L. [white jute] and *C. olitorius* L. [tossa jute]) and Mesta crops (*Hibiscus cannabinus* [kenaf] and *H. sabdariffa* [roselle]) were sap-inoculated, they were found to be symptomless carriers of the virus, and amazingly, the virus infection enhanced productivity in all these crops without any detrimental effect (Ghosh 1982).

RNMV is bipartite, having flexous particles of 275 × 13–14 nm and 550 × 13–14 nm (Inouye 1977; Ghosh 1982). The Indian isolate of RNMV is serologically related to the Japanese isolate, and they are also similar in morphology and size. The virus is transmitted to rice by a monocot-specific, soil-inhabiting fungus, *Polymixa graminis*, and to dicot hosts by sap. There is no known insect vector of RNMV (Ghosh 1980; Inouye and Fujii 1977). Initially, rice was the only known host, but later *Triticum aestivum*, *Avena sativa*, *Zea mays*, *Chenopodium amaranticolor*, *C. quinoa*, *Nicotiana tabacum*, *Brachiaria ramose*, *Ludwigia perennis*, and the aforementioned jute and mesta crops were also infected by RNMV (Ghosh 1981, 1982; Inouye and Fujii 1977).

Important fiber crops, jute and mesta are natural inhabitants of the tropics and subtropics. In India these crops are cultivated in several states such as West Bengal, Assam, Meghalaya, Tripura, Orissa, Bihar, Uttar Pradesh, Madhya Pradesh, Andhra Pradesh. Tossa jute originated in Africa and white jute in Indo-Burma region. In India tossa jute is called the "poor farmers' golden fiber" and is preferred over white jute for its higher productivity. For some time, polypropylene fibers captured the market over these centuries-old natural fibers because of their lower cost and better fiber strength. Recently, however, as we recognize that nonbiodegradable polypropylene fibers are a potential threat to our environment, the market for natural fibers has been revived.

Productivity promotion

As discussed before, RNMV-sap-inoculated jute and mesta seedlings were symptomless hosts of the virus and had enhanced productivity at full maturity (Ghosh 1982). In relation to the noninoculated control plants, virus infection improved the vascular system and increased plant biomass from 8 to 220 g with greater vigor and enhanced juvenility. Stem diameter, leaf and root size increased, and the crop required only half of the normally recommended dose of fertilizer as compared to noninoculated control crop (Ghosh and Mitra 1987). In a study of the RNMV–host interactions in jute and mesta, the inherent cytokinin-like activity in the plants was greatly enhanced while auxin-like

activity was retarded (Ghosh 1985). This RNMV-induced growth promotion in jute and mesta is maintained through harvested seeds for three generations (Ghosh 2001). Such seeds have been called energized (E) seeds.

Tetraploidy, is commonly used for crop improvement. In an investigation (Ghosh and Mitra 2008) of tetraploid tossa jute varieties JRO-1265 (4*n*) a progenitor of JRO-632, JRO-1055 (4n) a progenitor of JRO-878 and JRO 2020 (4*n*) a progenitor of JRO-3690 and diploid tossa JRO 3690 (2*n*), JRO 878, JRO-524 (2*n*), JRO-TJ-40 (2*n*) and JRO-36E (2*n*) (100 plants of each variety), 20 days after germination of seeds, the apical leaves of each plant were mechanically inoculated with sap from RNMV-infected rice leaves. Seeds were harvested at maturity (120 days), and productivity was analyzed; irrespective of ploidy, all the varieties except for JRD-878 (2*n*) increased in plant height, stem diameter, total fresh mass (with and without leaves), fiber mass and stick mass. However, the degree of the increase varied with variety. The fiber mass increased from 22.43 to 36.00% in diploids and in tetraploids from 2.80 to 19.00%. Similar interesting results were earlier seen with other varieties (Ghosh 1985). In these fiber plants, RNMV infection stimulates cell division and increases the cell size, contributing to the increases in leaf size, stem diameter and fiber bundle number (Ghosh 1982). RNMV infection at the seedling growth and a positive serological test at crop maturity, coupled with enhanced endogenous growth induced by cytokinin and IAA suggests that a unique pseudosymbiotic relationship between the host and virus induced beneficial biochemical changes (Ghosh 1985). Because tetraploidy is a better avenue for higher production in many crops, using RNMV to further improve phenotypic characters that increase yield potential in such tetraploids has opened a new vista that is less time consuming and cheaper to develop for commercial use (Ghosh and Mitra 2008). RNMV is not transmitted from jute to the succeeding rice crop in a rotation because the fungus vector *P. graminis* in soil is specific for monocots and thus makes the technology even more viable (Ghosh 1985).

Mesta yellow vein mosaic disease of mesta crops (jute and kneaf)

Mesta crops are an important source of bast fibers and a substitute for jute. The fibers obtained after retting are used as the raw material for a number of products such as rope, fishing net, and canvas. Seeds are a good source of oil (20%) and used in cooking, soap, paints and lubricants. Seed cakes left after oil extraction are used as cattle feed. The plant also has medicinal value against several human disorders such as coughs and rheumatism. The U.S. Deparment of Agriculture has identified kneaf as the best plant for making nonwoody paper.

Mests crops are grown in India in several states, but overall productivity of the crops is low. Viral diseases, most importantly Mesta yellow vein mosaic disease (MYVMD), are an important factor in reducing the fiber yield. MYVMD was first observed in Bahraich district in Uttar Pradesh and has spread fast in several other areas of the country, incurring heavy losses. The causal agent of the disease has been identified as monopartite begomovirus associated with satellite DNA β (Chatterjee et al. 2005; Paul et al. 2006). Southern blot and nucleic acid hybridization test using *Cotton leafcurl Rajasthan virus* (CLCuRV) specific DNA-A and DNA β probes confirmed that it is caused by a begomovirus related to CLCuRV. The disease is transmitted by cleft grafting and whitefly (*Bemisia tabaci*). Symptoms in emerging leaves start as abundant pin head spots on the leaf lamina, including veins. These spots gradually enlarge and coalesce to form chlorotic to yellow flecks. In severe cases, these yellow flecks enlarge until the leaves and stems are completely chlorotic or yellow and plant defoliates. Flower and pod set is reduced, those that develop are malformed. Life span of infected plants is also reduced. The host range of this virus differs from other begomovirus diseases on *Hibiscus* plants (Paul et al. 2006; Rajeshwari et al. 2005). The full-length sequence of DNA-A and DNA β of the virus infecting mesta was cloned and sequenced. DNA-A had 2728 nucleotides in length having 83.5% identity with *Cotton leafcurl Bangalore virus* (CLCuBV) and 83.3% identity with CLCuRV. There were six conserved ORFs identified in both orientations of the sequence. Sequence analyses proved that this new DNA-A was typical of Old World begomoviruses. It was therefore identified as a separate species of *Begomovirus* and was named as *Mesta yellow vein mosaic virus* (Chatterjee and Ghosh 2007).

Leaf fleck disease of sugarcane

Leaf fleck disease of sugarcane caused by *Sugarcane bacilliform virus* (SCBV) was first reported from Cuba (Rodríguez Lema et al. 1985). Later, Lockhart and Autrey (1988) reported that the sugarcane clone Mex. 57-473 growing in Morocco and Hawaii and CP44-101 in Morocco contained bacilliform viral particles, closely related to another identified *Badnavirus*, *Banana streak virus* (BSV) (Lockhart 1986; Lockhart and Olszewski 1993). The virus is transmitted by sugarcane mealy bug (*Saccharicoccus sacchari*) and the grey sugarcane mealy bug (*Dysmicoccus boninsis*) in nature, and citrus mealy bug (*Planococcus citri*), which does not colonize sugarcane naturally,

transmits SCBV experimentally (Lockhart and Autrey 1988; Lockhart et al. 1992).

In India the disease was first reported by Viswanathan et al. (1996). SCBV is now widespread in all sugarcane regions of the world with over 90% infection reported for noble cane germplasm (Comstock and Lockhart 1990). SCBV also infects *S. barberi, S. robustum, S. spontaneum, S. sinense* and commercial *Saccharum* sp. hybrids. The virus has also been detected in *Sorghum halepense*, *Brachiaria* sp., *Panicum maximum* and *Rotteboellia exaltata* (Lockhart et al. 1996). Although Autrey et al. (1995) suggested that SCBV originated in Papua New Guinea, the centre of origin of *Saccharum*, so far only commercial canes in Papua New Guinea have tested positive for SCBV, and these were introduced from Australia. Therefore, a more extensive survey of wild and commercial canes from Papua New Guinea and Indonesia, as well as India and China where other *Saccharum* species are thought to have originated may help elucidate the origin of the virus.

Badnaviruses have non-enveloped bacilliform particles measuring 130 × 30 nm and a dsDNA genome of 7.5–8.0 kb (Lockhart and Olszewski 1993). Badnaviruses resemble a second dsDNA genus, *Caulimovirus*, in the family *Caulimoviridae* and are para-retroviruses, which replicate via reverse transcription (Lockhart 1990). The DNA sequence of one isolate of SCBV has been published (Bouhida et al., 1993).

Symptoms of SCBV infection in sugarcane are variable and unreliable, and infected clones may remain symptomless (Autrey et al. 1995; Comstock and Lockhart 1990). Symptoms on noble canes vary from no symptoms to pronounced white, chlorotic flecks or streaks (Fig. 1e, f), while symptoms on commercial hybrids are generally less severe. SCBV is extremely widespread throughout noble cane germplasm, with reports of over 90% infection (Comstock and Lockhart 1990). There are two basic symptom types found using the world sugarcane germplasm collection at Cannanore, India (Viswanathan et al. 1996; Viswanathan and Premachandran 1998): (1) chlorotic specks that later turn to stripes and (2) mild mottling. More severe effects were observed on some clones, including stunted growth, fewer tillers, reduced and shorter internodes, bunchy top, and deep cracks on the internodes. Symptoms were very common on *S. officinarum*, and less common on *S. barberi, S. sinense, S. robustum* and interspecific hybrids. In many cases, clones with symptoms were found to be virus-free by ELISA or immunosorbent electron microscopy (ISEM), while, in a few cases, virus particles could be detected by ISEM in symptomless canes (Viswanathan and Premachandran 1998). A positive correlation between severity of symptoms and concentration of the virus was found only in some clones (Viswanathan et al. 1996).

The effect of SCBV on sugarcane yield has not been clearly determined. Circumstantial evidence suggests that SCBV contributed to decreased productivity in the clone Mex. 57-473 in Morocco (Lockhart and Autrey 1988). However, a field study to assess the effect of SCBV on sugarcane biomass production gave conflicting results (Comstock and Lockhart 1996). Strain variation, mixed infections, vector preferences and efficiency and environmental effects may all influence the effect SCBV has on sugarcane (Braithwaite 2001).

In India bacilliform virus particles of 126 × 30 nm were associated with the sugarcane filiform disease in the states of Uttar Pradesh, Maharashtra, Tamil Nadu and Haryana. Many of the symptomless leaf samples of the varieties had other clumps with freckling and yellow mosaic mottling symptoms also found to have bacilliform virus particles (126 × 30 nm) in leaf dip preparations (Singh et al. 2003). Incidence of the disease varied from 5–100%. The maximum incidence was recorded on variety Co 510 in Uttar Pradesh, Co 7219 in Maharashtra and Co P 94215, 97-28, 96-339, Co 89003 in Haryana. Positive serological reactions in DAC-ELISA and Western blotting were recorded with polyclonal antiserum of SCBV of a Mauritius isolate and BSV. Decoration of virus particle with SCBV and antiserum was also observed with Co S 510 sample during ISEM test. However, no relation was observed with the rice tungro virus antiserum. The symptomatology, particle morphology and serological relationship proved an association of SCBV with leaf fleck disease of sugarcane in India.

All the available methods for detecting SCBV have limitations. Diagnosis by symptomatology is unreliable, because many clones, particularly commercial hybrids, do not develop symptoms and the severity of symptoms may not be an indicator of virus titer. SCBV is not easily mechanically transmissible to common indicator hosts. Examination for virions in leaf dip preparation is suitable only in clones with high virus titer (Autrey et al. 1990; Singh et al. 2003) but at lower titer it can be improved by observing partially purified viral preparations (minipreps) under an electron microscope and by trapping viral particles with ISEM (Autrey et al. 1990; Singh et al. 2003).

The extensive genetic variation between SCBV isolates has also limited the usefulness and accuracy of DNA-based detection techniques. Dot-blots are unsuitable for detection because the cross-hybridization between DNA from genetically different isolates is very limited. This has been demonstrated by hybridizing labeled PCR viral DNA products with DNA amplified from different isolates. Distinct cross-hybridization groups were identified within the SCBV/BSV population (Lockhart and Olszewski 1993; Smith et al. 1996). RFLP patterns generated from digests of PCR products also revealed considerable variation (Smith et al. 1996). PCR can be modified to account for genetic

variation through the design of primers based on conserved sequences. Published primers for SCBV are available that target consensus sequences within the aspartic protease, ribonuclease H and reverse transcriptase coding regions and $tRNA^{Met}$ binding site of several badnaviruses (Braithwaite et al. 1995; Lockhart and Olszewski 1993). DNA sequencing can reveal the extent of genetic variation between isolates. When reverse transcriptase and ribonuclease H amplicons from SCBV isolated from three noble canes were sequenced (Braithwaite et al. 2004; Geijskes et al. 2002), variability was revealed not just among the SCBV isolates from different canes, but also among isolates within individual cane plants. Thus, conserved or degenerate primers may not reliably prime the amplification of all the isolates.

Sugarcane, particularly the noble canes, appear to contain a large pool of SCBV genomic sequences, perhaps because of the vegetative nature of sugarcane propagation and the long history of sugarcane movement and cultivation. Thus, SCBV variants have had considerable time to accumulate within the canes. The extensive isolate variability even within one sugarcane clone and the possibility of virus integration into the host genome have limited the acceptance of PCR-based methods for detection of SCBV. Although ISEM is time consuming and requires sophisticated equipment, it is still considered the most reliable method of detecting SCBV.

Control of the mealy bug vectors and disease-free planting material should be helpful in managing SCBV. Apical meristem culture and heat treatment could not eliminate SCBV from infected sugarcane (Autrey et al. 1990; Egeskov et al. 1994). Because badnaviruses replicate via reverse transcription, the use of chemotherapeutic agents that inhibit the reverse transcriptase offers a novel approach for control. Antiviral agents have been used successfully to eliminate BSV from banana (Helliot et al. 2003).

Banana bunchy top disease

Banana is one of the important fruit crops grown in tropical and subtropical areas of India, the world's largest banana producer. Although India contributes 19.71% of total world production (19.19 million tons) grown on 0.565 million hectares (Singh 2007), many pests of bananas and plantains diminish the productivity significantly, and viruses are considered a serious threat. So far, four viral diseases viz., bunchy top, streak, bract mosaic and infectious chlorosis have been reported in India. Of these virus diseases, banana bunchy top disease (BBTD) (Fig. 1g), caused by *Banana bunchy top virus* (BBTV) is the most serious and devastating disease, which limits or threatens production in one quarter of the world's banana-growing areas. Capoor and Varma (1968) reported mosaic disease of banana from Deccan area. Infectious chlorosis caused by *Cucumber mosaic virus* (CMV) is another important viral disease, more prevalent in Cavendish and Mysore group of bananas. Recently, banana bract mosaic and streak diseases reported at the farm of National Research Centre for Banana (NRCB), Tiruchirapalli (India) are assuming more significance owing to their fast spread and economic loss they cause (R. Selvarajan, personal communication).

BBTV is a single-stranded DNA virus in the family *Nanoviridae*. It is the type species of the genus *Babuvirus*. BBTV is isometric, and the particles measure 20 nm. The molecular weight of the coat protein is 19.3 kDa, and the virus has a multicomponent genome. Six circular single-stranded components are known to be associated with the genome (Selvarajan and Balasubramanian 2008). BBTV, introduced in India from Sri Lanka in 1943, has been listed under diseases of national importance. The disease has now spread to the states of Andhra Pradesh, Tamil Nadu, Orissa, Maharastra, Bihar, Karnataka, West Bengal, Assam and Uttar Pradesh and recently to the forests of North Eastern Hill states of India.

Banana bunchy top affects many commercial cultivars such as Virupakshi, Nendran, Robusta, Ney Poovan and Monthan, causing considerable economic loss, estimated at Rs. 400 million annually in Kerala alone. The highly flavoured hill banana, Virupakshi (AAB) has been wiped out by BBTV in lower Pulany and Sirumalai hills of Tamil Nadu (Selvarajan and Jeyabaskaran 2006). One of the reasons for the severity in hill regions is the year-round presence of inoculum and the insect vector that transmits the virus. The crop is grown perennially for 10–15 years as large clumps. The virus is transmitted primarily by infected suckers and through tissue-propagated material. Banana aphid *Pentalonia nigronervosa* transmits the virus in a semipersistent manner (Selvarajan et al. 2006).

Serological or immunological assays have been developed and used successfully for a number of years to detect plant viruses. Using polyclonal antiserum, DAC-ELISA was found better than DIBA for detection of BBTV. Simultaneous detection of Indian isolates of BBTV and BSV by duplex PCR and of BBTV, BBrMV and BSV by multiplex-PCR technique has been reported (Selvarajan et al. 2004). Selvarajan et al. (2004) developed IC-PCR technique for BBTV. Selvarajan et al. (2007) cloned and sequenced all six DNA components of a hill banana isolate of BBTV. The nucleotide sequences of BBTV-DNA 1 shared 99, 97 and 93% homology with the South Pacific group, the Asian group and the Indian group. BBTV-DNA 3 component had 96–97% homologies with the South Pacific and Asian groups, and BBTV-DNA 4 had 94–96%,

BBTV-DNA 5 had 94–99% and BBTV-DNA 6 had 97% homologies with the South Pacific group.

Selvarajan et al. (2007) reported cloning and sequencing of the coat protein gene of five BBTV isolates from the Arunachal Pradesh, Meghalaya, Nagaland, Assam and Kodaikanal hills. A comparative analysis of the Indian isolates with Asian isolates indicated that the Indian isolates do not belong to Asian group. Cluster analysis of the Indian isolate with South Pacific placed the Meghalaya isolate in a different cluster. A comparative analysis of all three groups (Asia, South Pacific and Indian isolates) further confirmed that the Indian isolates clusterd with the South Pacific isolates.

Banana bract mosaic disease

Bract mosaic (Fig. 1h), later established to be caused by *Banana bract mosaic virus* was first noticed in 1966 in India but as a malady of unknown etiology. Although this malady was later named Kokkan disease, a virus could not be associated with this disease. Sufficient time passed before the etiology could be established that the disease spread to unexpected levels not only in plantain Nendran, but also too many commercial cultivars such as Poovan, Robusta, Karpooravalli, Ney Poovan and Monthan. Presently, the virus is a major constraint in southern states of India; quarantine restriction is required to avoid further spread to other major banana-growing states. In Tamil Nadu, the percentage of BBrMV in Nendran, Poovan, Robusta, Red Banana, Ney Poovan, Monthan and Pisang Awak were, respectively, 15.93, 28.23, 4.57, 56.80, 5.31, 36.12 and 0.5% (Selvarajan and Singh 1997). Cooking bananas and Pisang Awak are also affected, but the yield loss is minimal. Selvarajan and Jeyabaskaran (2006) reported that the average yield reduction due to BBrMV was 30%. The reduction in bunch mass over healthy plants was 32.7, 53.69, 30.61 and 28.84% in four different fields, and the variations in bunch mass between infected and healthy plants were highly significant.

Banana bract mosaic disease (BBrMD) was first reported from Philippines in Cavendish varieties of banana (Espino et al. 1990). BBrMD is caused by *Banana bract mosaic virus* (BBrMV), which belongs to family *Potyviridae* and genus *Potyvirus*. Cytopathological observations revealed the presence of pinwheel inclusions and flexuous, rod-shaped particles (750 × 15 nm), typical features of the *Potyvirus* genus. The genome of the virus is positive-sense, single-stranded RNA of ~10 kb. The sequence of the C-terminal coding half of the CP and the 3′-untranslated region (3′ UTR) indicates that BBrMV is a distinct potyvirus. In India, the presence of BBrMV was confirmed on the basis of sequence homology and nucleic acid homology (Rodoni et al. 1997). BBrMD induces up to 70% yield loss in the southern Indian states (Cherian et al. 2002). Dark red, spindle-shaped streaks are seen on the bract of diseased banana plants (Fig. 1h). The emerging suckers also are dark red. Chlorotic streaks parallel to veins appear on the leaves. The PCR yielded a fragment of 900 nt for the CP-coding region and 162 nt for the 3′ UTR. The isolate had 98.1% and 99% homology with other Indian isolates, indicating a common origin (Sankaralingam et al. 2006). The virus titer is higher in the bracts and mid rib than in the leaf sheath. This virus is transmitted by *Rhopalosiphum maidi, Aphis gossypii* and *Pentalonia nigronervosa* in a nonpersistent manner. Presence of BBrMV can be detected by serology using ELISA (Selvarajan et al. 2006; Singh 2007) and has been detected from pseudostems and banana bracts using RT-PCR (Sankaralingam et al. 2006; Selvarajan et al. 2006).

NRCB has developed a dot blot technique for BBTV and BBrMV and other viruses of banana (R. Selvarajan, personal communication). This diagnostic technology is now used to test planting material and banana germplasm maintained at the centre. The NRCB has established state of the art facilities for banana virus research and diagnosis. The Molecular Virology lab has been accredited for virus indexing by the Department of Biotechnology and Department of Science and Technology, Government of India, under the National Certification System for Tissue Culture Raised Plants (NCS-TCP).

For developing transgenic bananas resistant to these viruses, BBTV and BSV have been characterized at the genomic level at NRCB. The BBTV coat protein and sense and antisense replicase gene constructs have been developed. This binary construct was mobilized from *E. coli* to *Agrobacterium tumefasciens* LBA4404 strain. It is designed to use all three constructs in the development of transgenics. The BBTV-DNA3 intergenic region with the *GusA* reporter gene is being assessed and compared for promoter activity. ECS development and regeneration protocols have been developed for Hill banana and Poovan bananas (R. Selvarajan, personal communication).

Citrus yellow mosaic disease

Association of badnavirus with citrus yellow mosaic disease (CYMD) was reported by Ahlawat et al. (1996). CYMD is widely prevalent in India in sweet orange (*Citrus sinensis* (L.) Osbeck) and pummelo (*Citrus grandis* (L.) Osbeck). The disease is transmissible by grafts and mealy bugs (*Planococcus citri*) (Ahlawat et al. 1996; Reddy 1997). CYMD is caused by *Citrus yellow mosaic virus* (CYMV) in the genus *Badnavirus*, family *Caulimoviridae*. CYNV particles are bacilliform, non-enveloped, contain

dsDNA (7559 bp) and measure 130 × 30 nm (Ahlawat et al. 1996; Huang and Hartung 2001). Infected citrus plants develop yellow mosaic (Fig. 1i) and fewer flowers and fruits. Juice and ascorbic acid levels are also reduced. Serological detection of badnaviruses is not reliable because the virus particles are weak immunogens. Ahlawat et al. (1996) obtained a PCR product using degenerate primers from partially purified virus preparations. However, the high level of polyphenolics and tannins in citrus leaves interfered in extracting good quality DNA. Baranwal et al. (2003) succeeded in overcoming this problem using sodium sulphite during the extraction. DNA thus obtained was higher in yield, better in quality and was cleaner and more stable. The sodium sulphite may have prevented oxidation of DNA upon release from host cells. This protocol has been standardized and is in regular use.

Piper yellow mottle disease

Betel vine (*Piper betle*), Indian long pepper (*Piper longum*) and black pepper (*P. nigrum*) are economically important species of family Piperaceae. After black pepper, one of the most ancient crops cultivated in India, in importance are betel vine and Indian long pepper. Black pepper may have originated in the southwestern hills of India, and betel vine is supposed to be a native of Malaysia. The medicinal properties of these plants are helpful against diseases of respiratory tract and are also important ingredients in the Ayurveda, Siddha and Unani systems of medicines (Ravindran 2000). Dried berries of *P. nigrum* (black pepper) are an important condiment of international commercial value and India earns Rs. 88 crore annually through export. Leaves of betel vine are chewed.

Piper yellow mottle disease caused by a badnavirus, piper yellow mottle virus (PYMoV), and transmitted by mealy bug (*Ferrisia* virgata) was reported in Southeast Asia including India (Bhat et al. 2003, 2005; Lockhart et al. 1997; Sarma et al. 2001). The symptoms on *P. betel* are mottling, mosaic and reduced leaf size; on *P. longum*, mosaic with dark green patches, blisters and leaf distortion; and on *P. nigrum*, vein clearing, mottling, chlorosis, and distortion of leaves, reduced plant vigor, and fewer and smaller fruit set. The virus on these hosts spread through the use of virus-contaminated plant material for vegetative propagation and via the mealy bug *F. virgata* and the citrus mealy bug *Planococcus citri* (Bhat et al. 2003, 2005).

Badnaviruses are non-enveloped bacilliform particles (30 × 130–150 nm), having a double-stranded, circular DNA genome (7.1–7.6 kb). The type member is *Commelina yellow mottle virus*. With a few exceptions, the genome has three open reading frames. ORF I and ORF II encode for putative proteins of unknown functions. ORF III encodes a polyprotein that is cleaved to produce functional products such as the movement protein, coat protein, aspartic protease and a replicase.

Siju et al. (2008) characterized badnavirus infecting betel vine and Indian long pepper. Degenerate primer pairs (sense and antisence) of ORF III were designed on the basis of known badnaviruses and used for PCR. The products were sequenced and were 597 nt long potentially coding for 199 amino acid sequence both in the case of betel vine and Indian long pepper. The sequence identities were 89.1% for nucleotides and 93.4% for amino acids when compared to those of known badnaviruses. The phylogenic tree clearly indicated that badnavirus isolates infecting betel vine and Indian long pepper and black pepper were strains of PYMoV.

Chrysanthemum diseases caused by *Chrysanthemum virus B* (carlavirus) and *Cucumber mosaic virus* (cucumovirus)

Chrysanthemum (*Dendranthema* × *grandiflorum* Kitam.) of family Asteraceae is a very popular cut flower crop and pot plant cultivated in several parts of India (4000 ha) and world. It is commonly known as garden chrysanthemum and "queen of the East." The word chrysanthemum comes from chrysos = golden and anthos = flower. It ranks only next to rose and third amongst the cut flowers in production in India (Singh 2000). The flower is most in demand in Japan and the Netherlands.

A large number of viruses and viroids viz. *Chrysanthemum virus B*, *Tomato aspermy virus*, *Cucumber mosaic virus*, *Tomato spotted wilt virus* (TSWV), *Chrysanthemum stem necrosis virus*, *Chrysanthemum spot potyvirus*, *Chrysanthemum chlorotic stunt viroid*, and *Chrysanthemum stunt viroid* are known to infect chrysanthemum in nature (Kumar et al. 2005, Singh et al. 2007a). In India, *Chrysanthemum virus B* (CVB), *Tomato aspermy virus* (TAV) and *Cucumber mosaic virus* (CMV) are most important (Verma et al. 2007). CVB is the most prevalent and does not cause any symptoms in most varieties of chrysanthemum. TAV and different strains of CMV induce significant losses. On some varieties, CVB causes vein clearing and mottling. It has narrow host range, confined to ~10 species of dicotyledonous plants. Chrysanthemum is vegetatively propagated by sucker, which aids disease perpetuation if the mother plant is virus contaminated. CVB is also transmitted mechanically and by *Myzus persicae* and *M. solani* (Hollings 1957). It is distantly related to carlaviruses viz. *Carnation latent virus*, *Potato virus S* and *Potato virus M* (van Slogteren et al. 1962). The host range of CVB does not overlap the host range of these carlaviruses.

Efficient diagnostic tools are important for screening the plant material and are essential for epidemiological studies, quarantine and effective marketing of virus-free plant material (Lawson 1981). Such tools are even more important for symptomless hosts. In India, protocols of diagnostic tools like ELISA, PCR, RT-PCR and IC-RT-PCR have been standardized for CVB (Singh et al. 2007a; Verma et al. 2003; Zaidi et al. 1993) and are now the routine for virus detection. In a further step, the coat protein (CP) gene, the triple gene block (TGB), the nucleic acid-binding protein (NABP), and Rep have been amplified, cloned and sequenced. The Rep gene was completely sequenced. The CP gene sequences of 29 isolates collected from different places showed variation in size (9921–969 bp); the middle portion of the CP was conserved but the N and C terminals varied markedly (Singh et al. 2007a). In phylogenetic analyses, the relationship of CVB to *Lily symptomless virus* was established. All 29 isolates were classified into three major groups in the phylogenic tree.

CMV induced symptom variability in chrysanthemum is common in nature. It may be yellow mosaic with green veins, severe mosaic, yellowing of veins and yellow to necrotic spots. This variability in symptoms is a strong indication of the existence of CMV strains in the natural infection of chrysanthemum (Srivastava et al. 1992). The virus particles in dip preparation of these samples of varied symptoms were isometric measuring 29 nm. Association of CMV with these varied symptoms was confirmed by RT-PCR. CP gene-specific primers of CMV gave a band of ~650 bp, which hybridized with the cloned probe of CMV (Kumar et al. 2005).

Chrysanthemum diseases caused by *Tomato aspermy virus* (cucumovirus)

Chlorotic ring mosaic of chrysanthemum caused by *Tomato aspermy virus* (TAV) in India is characterized by yellow mosaic, green vein banding, stunting and flower deformation (Fig. 1j, k). TAV has been identified on the basis of symptoms, host range and aphid transmission (*Aphis gossypii* and *Myzus persicae*) (Gupta and Singh 1981; Sastry 1964). Later, TAV was identified by other features: local lesion hosts (*Chenopodium* spp. and *Cucumis sativus*), electron microscopy (ca. 29 nm isometric particles), crystalline inclusions and virions in the central vacuole in cells of *N. clevelandii* after sap-inoculation, serological relationships, molecular weight of the CP, the presence of five RNAs (Raj et al. 1992, 2007; Verma et al. 2007). Verma et al. (2007) for the first time obtained complete amplification of the TAV genome (RNA 1, RNA 2, RNA 3) using multiplex PCR. Fifteen isolates of TAV on different cultivars of chrysanthemum were collected from different states of India, from the plains and from hilly tracts at higher elevations. Both in RT-PCR and IC-RT-PCR, an amplicon of the expected size (657 bp) was observed on an agarose gel. The virus could be detected by RT-PCR in plants without symptoms, but no virus was detected by ELISA in these samples. Amplified CP gene sequences of four isolates had 97% identity with those of other isolates available in the database, and the deduced amino acid sequence also had 99% similarity, probably indicating a common ancestry. The lower similarity between the CP genes of TAV and CMV indicated a distant relationship within the group. Some of the samples with a faint amplification of the 657-bp fragment in the RT-PCR resulted in a strong amplification in the IC-RT-PCR.

Gladiolus diseases caused by *Cucumber mosaic virus* (cucumovirus)

Among the top six flowers in the growing floral export industry in India, *Gladiolus psittacinus* (Iridaceae) is a very important cut flower widely used in bouquets and floral baskets because of its very attractive inflorescences in a wide range of colors. Numerous viruses infect gladioli in nature: CMV, *Bean yellow mosaic virus* (BYMV), *Broad bean wilt virus* (BBWV), TSWV, *Tobacco mosaic virus* (TMV), *Tobacco necrosis virus* (TobNV), *Tobacco black ring virus* (TobBRV), *Tobacco ring spot virus* (TobRSV), *Tomato black ring spot virus*, *Arabis mosaic virus*, *Tobacco streak virus*, *Soybean Mosaic virus* (SMV) and *Strawberry latent ring spot virus* (StrLRSV) (Katoch et al. 2003; Raj et al. 1998, 2002). Of these, CMV and BYMV are the most important viral pathogens of gladiolus. They produce mosaic, stripe, floral color breaking and reduced vigour (Fig. 1l, m). The virus is transmitted mechanically and by *M. persicae* in a nonpersistent manner. These viruses were identified on gladiolus on the basis of symptoms, the host range (which is wide), aphid transmission, electron microscopy, serological tests viz. DAS-ELISA, TAS-ELISA, direct tissue blotting immunoassay (DTBIA), ISEM, Western blotting assay (WBA), and RT-PCR and Southern hybridization. Of all these, RT-PCR followed by Southern hybridization test was found most effective. Katoch et al. (2003) reported that DAS-ELISA, DTBIA, ISEM and RT-PCR were best suited to detect these viruses. ELISA and DTBIA are most widely used, but the RT-PCR is more sensitive and can detect virus even at very low titer.

Important virus diseases of orchids in India

About 1300 species of orchids occur in different parts of India especially the northeastern states, northern hilly

areas, Kerala, and the Darjeeling hills. More than 30 viruses have been reported to infect different orchids in various geographical locations. Among the orchid viruses, *Cymbidium mosaic potexvirus* (CymMV) and *Odontoglossum ringspot tobamovirus* (ORSV) are considered most important due to their worldwide occurrence and severity of the symptoms in several orchid genera. They reduce the general vigor of the plant and affect the flower quality, thereby reducing the marketability and incurring severe economic losses. These two viruses also occur naturally as mixed infections in several orchid species. In India, almost no attention has been given to viral diseases of orchids because orchid growers have been ignorant about viral diseases and their economic implications on orchid plants.

Orchid viruses generally produce mosaic, ringspots, chlorosis, chlorotic and necrotic sunken patches on the leaves, deformation and a drastic reduction in the size of flowers and overall stunting of the plant. The most important symptoms of CymMV and ORSV on orchids are mosaic and mottling on the leaves, followed by necrosis of the leaves and flowering stalk. CymMV induces characteristic colour breaking in *Cattleya*. *Calanthe mild mosaic potyvirus* also induces flower color break in *Calanthe* (Gara et al. 1998).

Sherpa et al. (2003) first reported the occurrence of CymMV in India and recorded the virus in *Cymbidium aloifolium*, *C. iridioides*, *Epidendron* sp., *Liparis botanensis*, *Phaius tankervilleae* and *Pholidota imbricata* and cymbidium hybrids. The virus was successfully transmitted to *Datura stramonium* and *Chenopodium murale*, which produced blotchy local lesions. The presence of the virus in local lesions was confirmed by ELISA and electron microscopy. Later, Sherpa et al. (2006a) reported ORSV in 42 different species of orchids from Sikkim by slot-blot hybridization method. They then characterized the coat protein (CP) gene of CymMV from India and amplified the gene encoding the CP gene of nine isolates using the RT-PCR (Sherpa et al. 2006b). They also cloned and sequenced the amplified product, and multiple gene sequence alignments of the deduced amino acid sequence revealed considerable homology to CymMV from other countries. They concluded that CymMV CP gene is highly conserved and can be used for diagnosis. Bhat et al. (2006) reported CymMV in vanilla from the states of Kerala and Karnataka. Singh et al. (2007b) reported the association of a potyvirus with *C. pendulum* and *C. tigrinum* from Sikkim. The infected samples when tested by ELISA, RT-PCR and Northern blot analysis confirmed the presence of potyvirus. The sequencing of a RT-PCR amplified amplicon using potyvirus-specific primers revealed that the virus is closely related to *Calanthe mild mosaic virus*.

Recently, a large number of orchid germplasms was scanned by EM and ELISA at the National Research Centre for Orchids, Pakyong, Sikkim. Electron microscopy of a negatively stained preparation from the sap of mosaic-affected cymbidium and other orchid species showed flexuous particles measuring about 450–550 nm, while sap from ORSV-affected plants had rigid rods measuring about 300–310 nm. Electron microscopy and ELISA results revealed that mixed infections of CymMV and ORSV are common in nature; CymMV and ORSV the most common viruses in northeastern Himalayan region of India. Besides these two viruses, potyvirus-, rhabdovirus-, badnavirus- and tombusvirus-like particles have also been recorded in different orchid genera and species. Attempts are being made to characterize these viruses biologically, serologically and molecularly in order to develop effective strategies for producing healthy plant materials and thereby compete with other countries involved in the orchid trade (Pant et al. 2007).

In addition to the diseases of different crops discussed, a few more that are caused by begomoviruses are currently of significance in other crops; also *Sugarcane streak mosaic virus*, which is different from *Sugarcane mosaic virus* and has been recommended to be renamed as a new genus of *Potyviridae*; *Tobacco streak virus* of genus *Ilarvirus*, family *Bromoviridae*; *Mandarivirus*, a new genus of the newly created family *Flexiviridae*; and *Pigeonpea sterility mosaic virus* (unassigned). The diseases they cause viz. cotton leaf curl (Sharma and Rishi 2007, 2008); sugarcane streak (Hema et al. 2008); necrosis diseases of sunflower, legumes, cucurbits, chili, okra, cotton and sun hemp (Jain et al. 2008); Indian citrus ring spot (Ahlawat and Pant 2008); and pigeonpea sterility mosaic (Kumar et al. 2004) have been adequately dealt with in recent reviews (Rishi 2004, 2006).

Conclusion

India has a diverse topography, varying from fertile fields to the deserts of Rajasthan in the plains, a vast peninsular area, and the uplands of the Himalayas, Aravali and Nilgiri, that represent equally varied agroclimatic conditions from tropical, subtropical to temperate. These conditions have led to the evolution of one of the richest biodiversity of species in the world.

Indian agriculture is also one of the oldest in world. There is documented evidence of the export of spices, cotton and silk for centuries to different parts of the world. Tropical and subtropical agroclimates are conducive to the growth of a large number of pests and pathogens, which have coevolved with the rich vegetation. Symptoms of some of these biotic stresses such as leaf fleck disease of sugarcane are so obscure that they can long miss the attention of even highly experienced scientists and farmers.

Literature is replete with examples of the virus contamination of entire lots of germplasm and their movement and inadvertent spread to different parts of the world (Comstock and Lockhart 1990; Hampton et al. 1976; Lockhart et al. 1996; Viswanathan et al. 1996). These germplasms were long used for breeding purposes, which disseminated these viruses. Furthermore, during a long coexistence, part of the genome of some viral pathogens became integrated within the host as in sugarcane where propagation is vegetative (Braithwaite et al. 2004). In such cases, the results of PCR are deceptive, and ELISA/ISEM or Western blotting tests are more dependable.

In spite of the fact that India today is one of the five most vibrant economies in the world, Indian agriculture still forms the backbone of a rural livelihood and national economy. The rapid growth of agriculture to ensure food security and the liberalization of world trade in agriculture with the advent of the WTO have opened new vistas of growth while imposing new challenges with the risks of introducing new pathogens, existing pathogens adapting to new hosts, and discovering undeclared transgenicity in a sample. To meet these challenges, the National Bureau of Plant Genetic Resources, New Delhi is the mandatory nodal agency for the management of plant genetic resources including germplasm exchange. It has a Containment Level-4 facility for processing transgenics and a state of the art facility and the expertise to detect and intercept seed-transmitted viruses and virus-contaminated plant material. An international standard (http://www.ippc.org) for phytosanitary measures and other national regulatory measures to protect agriculture have been (Khetarpal and Gupta 2002, 2006) promulgated. A National Agricultural Biosecurity System should soon come to force and will integrate with the existing regulatory mechanisms under the auspices of various agencies (Khetarpal, personal communication).

During the last three decades, plant virology has rapidly developed with the advent of advanced molecular techniques that have led to the identification of a large number of viruses, thus strengthening the classification and taxonomy of plant viruses. Accurate and quick diagnosis of viral pathogens is imperative for the success of mapping of epidemics, breeding for resistance, and developing quarantine and other control measures. Problems with compounds such as polyphenolics, tannins, and polysaccharides that interfere with extracting good quality DNA have been overcome in several cases, and protocols for highly efficient, reliable diagnostics have been developed (Jose and Usha 2000). To tackle the multitude of problems in a vast country like India with diverse crops and vegetation, a few well-equipped laboratories of plant virology with the necessary expertise are functioning in some parts of India. The National Research Centre for Banana at Thiruchirapalli, Tamil Nadu and the Institute of Himalayan Bioresource and Technology at Palampur, Himachal Pradesh, have been accredited for virus indexing by the nodal departments of the Government of India, under the National Certification System for Tissue Culture Raised Plants (NCS-TCP). They also have developed state of the art plant virology laboratories for detecting, diagnosing and managing virus diseases and problems. But there is still need for several more such laboratories and additional centres for human resource development in the country. Ecofriendly measures such as marker-aided selection of disease resistance as is being developed for transgenic pigeonpeas resistant to pigeonpea sterility mosaic virus at ICRISAT, Hyderabad, pathogen-derived resistance, antiviral agents (Helliot et al. 2003), and biocontrol of insect vectors should be tried vigorously at much larger scales for long-term disease management.

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