1. Introduction

Bamboo plants are frequently infected by a wide range of pathogens (bacteria, fungi, viruses, etc.) that cause diseases of the rhizomes, roots, culms, foliage, flowers or seeds and are responsible for losses in yield and quality all over the world. Out of various pathogens infecting bamboos, those which affect the culms are recognized as most significant ones as they cause extensive damage to the bamboo plants (Mohanan, 2002). Some of the potentially serious diseases of bamboos include fungal diseases like culm blight, culm rot, culm rust and witches broom, small leaf disease and culm mosaic (Mohanan, 2002). While there is an array of fungal and bacterial diseases known to infect bamboos and their etiology is also well documented, very little is known about the viral diseases infecting bamboos.

Plant viruses are infectious, intracellular, obligate parasites comprising RNA or DNA genomes surrounded by a virus-encoded protein coat, assembled in rod shaped or isometric spherical particles that infect plant cells, altering their chemistry and causing a wide range of disease symptoms. Currently, there are around 1,016 known plant viruses and based upon the properties of their virions, antigenic and biological properties, genome organization and replication, these viruses have been placed into three orders, 21 families and 92 genera (10 are unassigned) by the International Committee on Taxonomy of Viruses (ICTV) (Hull, 2013). Apart from the viral diseases, plants have been known to be infected with various economically significant virus-like diseases which are caused by pathogenic RNAs known as viroids. They are small, single stranded, circular molecules (200-400 nucleotides) having high degree of self complementarily, that do not code for any protein and replicate independently of the associated virus. Based on the sequence and predicted secondary structure of their RNAs, viroids are classified into two families Pospiviroideae and Asunviroideae (Hull, 2009).
Plant viruses need a living host for their perpetuation and gain entry into the host plant through wounds, mechanical injury, insect vectors, methods employed in pruning and vegetative propagation, etc. whereas some viruses are seed- or pollen-borne. In order to complete their life-cycle within the host plant, viruses evade host defence system and take over the functions of different host factors altering the cellular processes and normal functioning of the host cell, thereby, causing disease symptoms. Depending upon the type of virus, host plant and environmental conditions, viruses may or may not induce noticeable disease. While latent infection by a virus remains mostly unnoticed, problem arises when such hosts act as reservoir of the pathogen and by transmitting the virus to other plants, lead to severe symptoms and even death of the susceptible host plants (Mathews, 2010).

Virtually, all plants are infected by viruses but those from cultivated food crops are the most studied because of the economic impact of the losses associated with them. However, it is important to acknowledge that all plants that are grown for fodder, fibre or wood are also hosts to many viruses which may not have immediate effect but a significant indirect effect due to the damage caused to the plants, thus, leading to economic, agronomic and social impact (Hull, 2009).

Various newly emerging virus problems in crops are being driven mainly due to changes in agricultural practices, global trade and the climate. Viral diseases are usually less pronounced and generally last for a lifetime (Hull, 2013). This is particularly true for perennial crops and those that are propagated by vegetative means. The methods used for the vegetative propagation of plants like grafting, cutting, budding, etc. have been recognized as the most important ways for perpetuation of virus and virus-like diseases. Bamboos are routinely multiplied by vegetative propagation and hence act as source of infection for the progeny (Mohanan, 2002).

There is a preliminary evidence that at least one of the diseases caused by Cherry necrotic rusty mottle virus (CNRMV) in bamboos, may also be spread by insect vectors such as aphids or delphacids.

2. Viral Diseases

2.1. Bamboo Mosaic Virus (BaMV)

It was the first virus to be identified infecting bamboo plants. The virus was first isolated from Bambusa multiplex (Lour) Raeusch. and B. vulgaris Schrad. and reported from Brazil in 1975 (Kitajima et al., 1975; Lin et al., 1977). It has also been reported from Taiwan (with a disease incidence of about 70-80%), California and Florida (USA), Australia and Hawaii (Lin et al., 1979; Lin and Chen, 1991; Lin et al.,
2.1.1. Causal organism

*Bamboo mosaic virus* belongs to the genus *Potexvirus* in the family *Alphaflexiviridae* (Hull, 2013). BaMV particles are flexuous, filamentous rods, 480-500 nm in length and 15 nm in diameter (Lin *et al.*, 1977). The virus is thermally inactivated between 75°C to 80°C and loses infectivity when diluted to 10⁶. The virus is infectious when stored at 24°C for one month or at -15°C for four months (Chen, 1985).

Under the electron microscope, the virions of BaMV can be seen in epidermis, mesophyll and vascular bundles of the infected tissues (Lin and Chen, 1991). Within the infected cells, the virus appears in the form of electron dense crystalline bodies (EDCBs) with varying shapes and sizes (0.1 to 2.7 µm) and aggregates of virions in the chloroplast, cytoplasm, vacuoles and the nucleus. Within these organelles, the virions are arranged in loose parallel or helical fashion and can also be seen irregularly distributed in the cytoplasm. The precise location of virions and the EDCBs within the infected cells can indicate the stage of infection (Lin and Chen, 1991; Chang *et al.*, 1997). Virions can be purified by extraction in 0.5 M borate buffer (added with 1mM EDTA) at pH 9 with Triton x 100 and PEG 6000, followed by differential centrifugation and CsCl density gradient separation (Lin and Chen, 1991).

The genome of BaMV is monopartite, comprising of ~6.4 kb positive sense RNA with 28kDa capsid protein subunits (Lin *et al.*, 1992). In some variants, the genomic RNA of BaMV is also known to be associated with two subgenomic RNAs (2.0 and 1.0 Kb in size) which are encapsidated within the coat protein of the virus and probably responsible for expression of the ORF2 protein and capsid protein, respectively (Lin *et al.*, 1992).

Some isolates of the virus, also contain a satellite RNA (sat BaMV) which is a single stranded RNA sub-viral agent whose replication is supported by *Bamboo mosaic virus*, encapsidated by BaMV capsid protein to form rod shaped particles of length 60 nm (Lin and Hsu, 1994). SatBaMV was first identified to be associated with BaMV isolated from *B. vulgaris* McClure and reported from Taiwan in 1994 (Lin and Hsu, 1994). SatBaMV particles can co-purify with BaMV virions by centrifugation in a CsCl gradient which can be separated from BaMV particles by centrifugation in a 10-40 per cent sucrose density gradient (Lin and Hsu, 1994).

2.1.2. Genome organization

The genome of BaMV is comprised of a single-stranded, positive-sense RNA, 6,366 nucleotides, with a 5' cap structure and 3' poly (A) tail and six conserved open reading
frames (ORFs 1 to 6) (Fig. 2.1.2.1.) (Lin and Hsu, 1994). ORF1 encodes a 155kDa protein which is responsible for RNA replication and has domains with methyltransferase, helicase and polymerase activity. The overlapping ORFs 2, 3 and 4, constitute the triple gene block, and code for viral movement proteins of 28, 13 and 6 kDa, respectively. ORF5 codes for 25 kDa capsid protein of the virus. The ORF 6 which lies completely within ORF1 distinguishes the genome organization of BaMV from that of other potexviruses sequenced so far. The putative product of ORF6 (14 kDa) shows no significant similarity to the products encoded by the ORFs of the other known potexvirus (Lin and Hsu, 1994).

SatBaMv is a linear RNA molecule of 836 nucleotides, having cap structure at its 5' end and a poly (A) tail at its 3' end. It consists of an ORF which codes for a 20kDa protein (183 amino acids) flanked by 5' and 3' non-coding regions of 159nt and 129nt, respectively. The protein p20 is a RNA binding protein which helps in the systemic movement of the SatBaMV in the co-infected plants but is not essential for its replication (Lin and Hsu, 1994; Lin et al., 1996; Palani et al., 2012). The nucleotide sequence of this protein (p20) is also highly conserved among all variants of SatBaMV.

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Fig. 2.1.2.1. Diagrammatic representation of the genome organization of BaMV.

2.1.3. Symptoms

Characteristic symptoms caused by this virus include foliar mosaic and stripe, brown internal streaking of the shoots and culms, and culm abortion (Fig. 2.1.3.1.) (Lin and Chen, 1991; Elliot and Zettler, 1996). Culms are poorly developed with shortened internodes and the newly emerging shoots are hard in texture, thereby, depleting their quality for eating and canning (Lin and Chen, 1991). For these reasons, Bamboo mosaic virus is being considered as a limiting factor in the production of edible bamboos in Taiwan (Lin and Chen, 1991; Hsu et al., 2000).

Some variants of SatBaMV (BSL6 isolated from D. latiflorus Munro) strongly interfere with BaMV replication and attenuate the symptom caused by BaMV infection (Hsu et al., 2006; Chen et al., 2007; Chen et al., 2012). Some isolates like BSF4, first isolated from B. vulgaris, do not have any significant effect on BaMV (Lin and Hsu, 1994; Lin et al., 1996; Hsu et al., 1998). Genetic studies have shown that various mutations at the 5’ apical hairpin stem loop (AHSL) region affect the
ability of SatBaMV to interfere with the replication of its helper virus (Chen et al., 2007; Chen et al., 2012).

2.1.4. Host range
So far BaMV is known to infect bamboos exclusively with no other known natural host. This virus has been known to infect 13 species of bamboos (mainly those with pachymorph rhizomes) namely: B. mutabilis, B. beecheyana, B. dolichoclada, B. edulis, B. multiplex, B. oldhamii, B. pachinensis, B. utilis, B. ventricosa, B. vulgaris, D. giganteous, D. latiflorus and Melocanna baccifera (Kitajima et al., 1975; Kitajima et al., 1977; Lin et al., 1977; Chen, 1985; Lin and Chen, 1991; Lin et al., 1992; Lin et al., 1993; Elliot and Zettler, 1996; Chang et al., 1997; Lee et al., 1998; Dodman and Thomas, 1999).

Experimentally BaMV can be transmitted mechanically (0.02M sodium phosphate buffer pH 7.2) to B. vulgaris 'vittatu', Nicotiana benthamiana, Chenopodium amaranticolor, C. ficiolium, C. murale, C. quinoa, C. occidentalis, Gomphrena globosa and Hordeum vulgare (Chen, 1985; Lin and Hsu, 1994; Elliot and Zettler, 1996; Chang et al., 1997; Lee et al., 1998).

SatBaMV can be transmitted mechanically, along with the helper BaMV to, Chenopodium, Hordeum, and N. benthamiana Domin but is known not to be transmitted to other Nicotiana sp., Phaseolus or Triticum (Lin and Hsu, 1994).

2.1.5. Transmission
BaMV has no known insect vectors. Bamboos are normally vegetatively propagated and the virus is probably transmitted through vegetative propagation of infected, non-indexed planting material and mechanically by the unclean practices employed in the routine cutting of bamboo shoots (Lin and Chen, 1991).

2.1.6. Diagnosis and identification
BaMV and its satellite RNA (SatBaMV) can be detected by serological tests like ELISA, nucleic acid hybridization using radiolabelled or non-radiolabelled probes or RT-PCR (Lin and Hsu, 1994; Hsu et al., 2000). Alternatively, virus can be inoculated on assay host like G. globosa and C. amaranticolor (necrotic local lesions); B. vulgaris cv. Vittatu (W) and D. latiflorus cv. Mei-nung (W) (systemic mosaic) for symptom development (Dallwitz, 1980; Dallwitz et al., 1993; Bruntt et al., 1996; Nelson and Borth, 2011).

2.1.7. Control measures
Once established, the disease caused by Bamboo mosaic virus cannot be eradicated without destroying the infected plants. Therefore, the use of disease-management
practices is the only means to prevent further spread of the infection. The infected stock should be isolated and only virus free planting material be used for further propagation of the nursery trees. Meristem tip culture technique is being used for the production of virus free bamboos, but for such plants also, it is necessary to know whether the material in virus free or not for further culturing. Pruning of diseased plants should be done carefully and blades must be sterilized between each use to minimize dissemination of the disease from infected to the healthy plants (Hsu et al., 2000; Nelson and Borth, 2011).

2.2. Cherry Necrotic Rusty Mottle Virus (CNRMV)

*Cherry necrotic rusty mottle virus* has mainly been associated with a disease of sweet cherries. The first incidence of the disease was described in 1945 in Utah (Rhoads, 1945) and since then the virus has been reported from many cherry growing regions of the world like Chile, China, Japan, Europe, Germany, India, Korea, New Zealand and North America (Wadley and Nyland, 1976; Rott and Jelkmann, 2001a; Gentit et al., 2002; Isogai et al., 2004; Mandic et al., 2007; Fiore and Zamorano, 2013; Noorani et al., 2013; Zhou et al., 2013; Cho et al., 2014).

2.2.1. Causal organism

*Cherry necrotic rusty mottle virus* (CNRMV) is an unassigned member of the family *Betaflexiviridae*, flexuous filamentous plant viruses (Adams et al., 2012). Not much is known about their physical or chemical properties.

2.2.2. Genome organization

The genome of CNRMV comprises of a single-stranded, positive sense RNA ~8.4 kb in size (excluding the 3'poly (A) tail) which consists of seven open reading frames (ORFs) (Fig. 2.2.2.1.) (Rott and Jelkmann, 2001a). Five of these ORFs (1 to 5) are conserved among all fovea-, allexi-, potex- and carlaviruses and code for the replicase (ORF1), the triple gene block (TGB) movement proteins (ORF 2, 3 and 4) and the coat protein (ORF 5). Two other ORFs, of unknown function, ORFs 2a and 5a, are nested completely within ORFs 2 and 5, respectively and the nucleotide sequence of these two ORFs is also not conserved among other related viruses (Rott and Jelkmann, 2001a).

![Fig. 2.2.2.1. Diagrammatic representation of the genome organization of CNRMV.](image-url)
2.2.3. Symptoms

The brown, angular, necrotic spots, abnormal colours and pattern, abnormal leaf fall, yellowing, rusty chlorotic areas, shot holes of the leaves (Wadley and Nyland, 1976); blisters, gummosis, resinosis, canker on woody stem; dieback and general necrosis of the bark; dieback of growing points and early senescence of the whole plant constitute the most important diagnostic symptoms of the disease (Richards and Reeves, 1951; Wadley, 1966). Disease symptoms are most severe in cultivars like Lambert, Seneca, Sam, Hudson and Bing, and moderate in Napoleon, Black Republican, Van, Windsor, Lyons, Macmar, Chinook and Rainier (Richards and Reeves, 1951; Wadley, 1959, 1966), while the symptoms may be very mild or masked in Black Tartarian, Burbank, Orb, Schmidt, Napa Long Stem Bing, Deacon, Cardofer Frühe and Dicke Braune Blankenburger cultivars (Posnette and Cropley, 1964).

The symptoms of CNRMV on bamboo plants constitute mosaic, chlorosis, yellow streaks, necrotic spots and curling on the foliage of infected plants (Fig. 2.2.3.1.) (Awasthi et al., 2014), while the effects of this virus on the growth and production of stem, pulp, shoot and rhizome of bamboos are yet to be determined.

2.2.4. Host range


2.2.5. Transmission

The disease caused by CNRMV is easily transmitted by grafting and budding but not mechanically by sap inoculation. There is no evidence of seed or pollen transmission of CNRMV (Rott and Jelkmann, 2012). In addition to graft transmission, natural spread of the disease has been observed in sweet cherry orchards in Oregon (Cameron and Moore, 1985), Utah (Wadley and Nyland, 1976), Montana (Afanasiev and Mills, 1957) and amongst bamboo clumps in India (Awasthi et al., 2014). CNRMV has been detected in two aphid species and in a delphacid (Unpublished data of Awasthi et al.) which might be acting as virus vectors.
Fig. 2.1.3.1. Symptoms caused by Bamboo mosaic virus. (a) Bamboo clump infected with Bamboo mosaic virus and (b and c) infected Bambusa vulgaris displaying distinctive interveinal chlorotic mosaic patterns and striping on the leaf surfaces (adapted with permission from Scott Nelson, University of Hawaii, USA).

Fig. 2.2.3.1. Virus like symptoms as seen on CNRMV infected bamboos. (a) Mosaic, chlorosis and yellow streaks on a leaf of D. hamiltonii (Local maggar) and (b) mosaic, chlorosis and yellow streaks on a leaf of D. hamiltonii (North East variety) (Awasthi et al., 2014).
2.2.6. Diagnosis and identification

Earlier methods used for the detection of CNRMV were mainly based upon graft indexing on the sweet cherry woody indicator 'Sam' (P. avium L.) (Diekmann and Putter 1996; Li and Mock, 2005). The recent methods developed for the detection of this virus include serological tests like DAS-ELISA and PAS-ELISA (Noorani et al., 2013; Zhou et al., 2013), nucleic acid hybridization using radiolabelled or non-radiolabelled probes (Awasthi et al., 2014) or RT-PCR based methods like degenerate oligonucleotide primed-PCR (DOP-PCR) (Rott and Jelkmann, 2001b); the polyvalent degenerated oligonucleotide nested RT-PCR (PDO nested RT-PCR) (Foissac et al., 2002); plate trapping (PT)-RT-PCR (Li and Mock, 2005) and high resolution melt (HRM) analysis (Komorowska, 2012).

2.2.7. Management

The disease caused by CNRMV can be managed by using certified virus-tested propagation material for raising new clumps and removing diseased culms/clumps as soon as diagnosed. When a large number of clumps are affected, new plantation should be set up.

3. Conclusion

Bamboos form the backbone of rural economy of many Asian countries and are now increasingly grown as plantation crops. However, the production potential of bamboos is greatly hampered by various biotic and abiotic factors which include pests and diseases. In addition to the fungal and bacterial diseases, newly discovered virus and virus-like diseases may emerge as serious threat to bamboo plantations. Unlike the bacterial or fungal diseases, the viral diseases cannot be eliminated from the infected plant and remain for a lifetime. There are even more serious consequences when the infected planting material is used for further propagation of nursery plants. Increasing knowledge of virus and virus-like diseases of bamboos necessitates the adoption of preventive measures. The most important preventive method seems to be production and use of virus free propagating material. A few viruses which are implicated in the diseases of commercial pome and stone fruit trees, have found alternate host amongst the bamboo plants. Though the information on these emerging viral diseases of bamboos is very preliminary and lacks details with regard to their impact on bamboo production, the findings indicate that these bamboo plants, besides themselves developing disease symptoms, may also be acting as source of infection for the stone and pome fruit trees. It would be important to understand the influence of insect vector populations on the distribution and spread of these viruses among other plant species.
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