Biology, Etiology, and Control of Virus Diseases of Banana and Plantain

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Contents

Abstract

Banana and plantain (*Musa* spp.), produced in 10.3 million ha in the tropics, are among the world's top 10 food crops. They are vegetatively propagated using suckers or tissue culture plants and grown almost as perennial plantations. These are prone to the accumulation of pests and pathogens, especially viruses which contribute to yield reduction and are also barriers to the international exchange of germplasm. The most economically important viruses of banana and plantain are *Banana bunchy top virus* (BBTV), a complex of banana streak viruses (BSVs) and *Banana bract mosaic virus* (BBrMV). BBTV is known to cause the most serious economic losses in the "Old World," contributing to a yield reduction of up to 100% and responsible for a dramatic reduction in cropping area. The BSVs exist as episomal and endogenous forms are known to be worldwide in distribution. In India and the Philippines, BBrMV is known to be economically important

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but recently the virus was discovered in Colombia and Costa Rica, thus signaling its spread into the "New World." Banana and plantain are also known to be susceptible to five other viruses of minor significance, such as *Abaca mosaic virus*, *Abaca bunchy top virus*, *Banana mild mosaic virus*, *Banana virus X*, and *Cucumber mosaic virus*. Studies over the past 100 years have contributed to important knowledge on disease biology, distribution, and spread. Research during the last 25 years have led to a better understanding of the virus–vector–host interactions, virus diversity, disease etiology, and epidemiology. In addition, new diagnostic tools were developed which were used for surveillance and the certification of planting material. Due to a lack of durable host resistance in the *Musa* spp., phytosanitary measures and the use of virus-free planting material are the major methods of virus control. The state of knowledge on BBTV, BBrMV, and BSVs, and other minor viruses, disease spread, and control are summarized in this review.

1. INTRODUCTION

Banana and plantain in the genus *Musa* (*Musicale*, Zingiberales) are among the major food crops and important horticultural crops produced in over 130 countries. They provide staple food and income for millions of smallholder farmers across the tropics in Africa, Asia, America, Oceania, and the Pacific (Fig. 1; Table 1). Total production was estimated at 139 million tons from 10.3 million ha in 2012; the two crops together rank no. 6 after maize, rice, wheat, potato, and cassava (FAOStat, 2014). All widely cultivated varieties are parthenocarpic, resulting from intra- and intercrosses of the two seedy species, *M. acuminata* ($2n = 2\times = AA$) and *M. balbisiana* ($2n = 2\times = BB$) (Ortiz, 2013). Many of the domesticated cultivars are natural mutants with triploid genome ($2n = 3\times = 33$) such as dessert banana (AAA), cooking banana, and plantain (AAB or ABB), and seedless cultivars of both diploids (AA and AB) and synthetic tetraploids ($2n = 4\times = 44$) with genome constitutions of AAAA, AAAB, AABB, and ABBB (Heslop-Harrison & Schwarzacher, 2007). Human migration and trade, especially in the Asia-Pacific, have played a major role in the wide dissemination of cultivars into Africa and Latin America (De Langhe & De Maret, 1999; Perrier et al., 2011). Currently over 1000 *Musa* accessions with a wide range of morphological traits and genome constitutions have been identified in collections worldwide (Ortiz, 2013; Pollefeys, Sharrock, & Arnaud, 2004).

Botanical distinction between banana and plantain is not straightforward. In general, banana are eaten uncooked as a dessert, and unripe starchy fruits

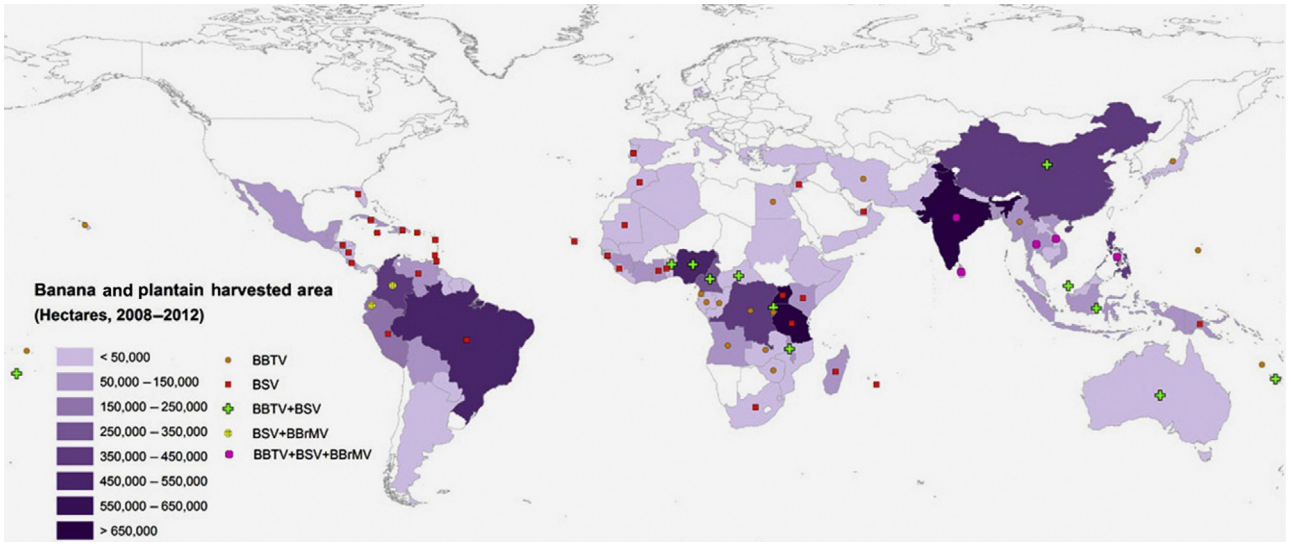

Figure 1 Banana and plantain production in various countries and distribution of the three major banana viruses, BBTV, BSVs, and BBrMV. (Average production data for banana and plantain for the years 2006–08. http://faostat.fao.org/)

Table 1 Combined area, production, and productivity of banana and plantain in 2012 and production trend from 2002–2012

	Area		Production		Yield	
	ha (×1000)	% Change	t (×1000)	% Change	t/ha	% Change
World	10,360.7	7.8	139,154.9	27.0	13.4	20.8
Africa	5862.2	4.1	42,408.1	20.3	7.2	16.9
Americas	2133.1	0.6	36,089.2	14.1	16.9	13.6
Asia	2258.0	24.2	58,731.0	40.3	26.0	21.3
Europe	10.5	0.8	399.9	−11.2	38.2	−12.1
Oceania	97.0	12.3	1526.7	19.4	15.7	8.1

% Change = Percentage increase or decrease compared with 2002 data.
Data source: FAO banana and plantain production statistics for 2012 (FAOStat, 2014).

that are cooked and eaten are referred to as plantain and cooking banana. Other cultivars are "beer banana" used for fermentation of the juice (Heslop-Harrison & Schwarzacher, 2007). Dessert banana are a major export commodity and a significant GDP earner for Ecuador, Costa Rica, Cameroon, and several other countries (FAOStat, 2012). Cooking banana and plantain are the most important sources of food and rural income in SSA (Nweke, Njoku, & Wilson, 1988). With 56.5% of the global area, Africa dominates the global *Musa* production area compared with 21.7% in Asia and 20.5% in Latin America. However, in terms of crop production and productivity, Africa is last, after Asia and America (Table 1). Dessert banana are grown mainly for commercial trade in Latin America and the Caribbean; cooking banana are widely grown in Asia-Pacific; plantain are dominant in Central and West Africa and Latin America; whereas the highland banana are mainly produced in East Africa (Ortiz & Swennen, 2014).

Banana and plantain grow, mature, and fruit without seasonality throughout the year. Suckers spring up from the underground rhizome to replace the main shoot that withers after fruiting, and this process of succession continues indefinitely (Morton, 1987). However, this exposes plantations to the effects of adverse environmental factors, pests, and pathogens. Farmers generally use young suckers removed from the old plantations to establish new fields. This practice has been among the major causes of outbreaks of several banana diseases and pests around the world (Jones, 2002), especially viruses, which are perpetuated along with the planting material.

About 20 virus species belonging to five families have been reported to infect banana and plantain worldwide (Fig. 1). *Banana bunchy top virus* (BBTV, genus *Babuvirus*, family *Nanoviridae*); several species of Banana streak viruses (BSVs, genus *Badnavirus*, family *Caulimoviridae*) are responsible for streak disease; and *Banana bract mosaic virus* (BBrMV, genus *Potyvirus*, family *Potyviridae*) are the most economically important viruses. *Abaca bunchy top virus* (ABTV, genus *Babuvirus*), abaca mosaic disease caused by a distinct strain of *Sugarcane mosaic virus* (SCMV) designated as SCMV-Ab (genus *Potyvirus*), *Banana mild mosaic virus*, and *Banana virus X* (BVX) both were unassigned members in the family *Betaflexiviridae*, and *Cucumber mosaic virus* (CMV, genus *Cucumovirus*, family *Bromoviridae*) are of minor significance. Viruses in general are a major concern to production because of their effects on yield and quality, and as constraints to the international exchange of *Musa* germplasm. Direct losses are incurred from reduced production, and indirect losses are associated with maintaining plant health, including the production of virus-free planting material. This review summarizes the present status of knowledge on the distribution, diversity diagnostics, and control of banana virus diseases.

2. MAJOR VIRUS DISEASES OF BANANA AND PLANTAIN

2.1. Banana bunchy top disease

2.1.1 Disease discovery and biology

Banana bunchy top disease (BBTD) is the most devastating virus disease of banana and plantain and is prevalent in the Old World (Dale, 1987). The disease was first recorded during an epidemic in Cavendish banana (AAA) in 1889 in Fiji (Magee, 1927). The origin of the virus in Fiji epidemic is not clearly known; however, it is supposed to have been introduced through infected suckers from Tanna (Vanuatu) (Simmonds, 1931). Available records indicate the wide dissemination of BBTD in the Old World along with the movement of planting material by humans, traders and returning soldiers in the early part of the twentieth century (Fahmy, 1927; Magee, 1927, 1953; Wardlaw, 1961). At present, BBTD occurs in 36 countries in Africa, Asia, and Oceania (Blomme et al., 2013; Diekmann & Putter, 1996; Jones, 2013; Kumar et al., 2011). Except for Hawaii (USA) (Conant, 1992) there are no records of BBTD in the New World.

Based on the symptoms, cytology, observation and features of vector transmission, the causal agent was initially established as a virus (Magee, 1940). Based on its localization within phloem tissue and the persistent mode

of transmission by *Pentalonia nigronervosa* Coquerel (Hemiptera, *Aphididae*), the putative virus in the BBTD etiology was suspected to be a member of the luteovirus group (Mathews, 1982; Wu & Su, 1990a). In 1991, studies (Harding, Burns, & Dale, 1991; Iskra, Garnier, & Bove, 1989; Thomas & Dietzgen, 1991) reported the purification of virus particles of 18 nm diameter consisting of a coat protein of 20.5 kDa and a single-stranded DNA of about 1 kb from infected plants. Polyclonal and monoclonal antibodies produced against the virus and nucleic acid probes reacted with extracts from symptomatic plants indicating their involvement in disease etiology (Thomas & Dietzgen, 1991). Further studies by Burns, Harding, and Dale (1995) identified additional five circular ssDNA components separately encapsidated. The virus, BBTV, is currently the type member of the genus *Babuvirus*, family *Nanoviridae* (King, Adams, Lefkowitz, & Carstens, 2012). It contains at least six ssDNA components, each about 1.1 kbp encoding for a single open reading frame (ORF) in the virion sense strand designated as: DNA-R (rolling-circle replication initiation protein), -S (coat protein peptide of 19.6 kDa), -M (movement protein), -C (cell cycle link protein), -N (nuclear shuttle protein), and -U3 (a protein of unknown function) (Beetham, Harding, & Dale, 1999; Burns et al., 1995; Vetten et al., 2012). A few BBTV isolates, especially in the East Asian region, carry a satellite DNA component that encodes a protein homologous to Rep encoded in DNA-R (Horser, Karan, Harding, & Dale, 2001).

2.1.2 Symptoms and economic importance

BBTV induces characteristic discontinuous dark green flecks and streaks of variable length on the leaf sheath, midrib, leaf veins, and petioles (Fig. 2). New leaves emerging from the infected plants are narrower with wavy leaf lamina and yellow leaf margins (Nelson, 2004). Leaves produced are progressively shorter, narrow, and brittle in texture; these bunch together at the top and hence provide the name of the disease (Thomas, Iskra-Caruana, & Jones, 1994). Susceptible cultivars infected at a young stage and the suckers emerging from infected stools are severely stunted. Severely infected plants usually will not fruit, but if fruit is produced, the hands and fingers are likely to be distorted and twisted (Nelson, 2004). Occasionally, bracts of male flower buds turn to a leafy structure and exhibit dark green dots and streaks (Thomas et al., 1994). Emerging suckers from infected plants exhibit severe symptoms. Plants infected at a later stage do not normally show leaf symptoms, but dark green streaks can be seen on the tips of the bracts. Emerging suckers from such plants usually exhibit moderate symptoms or none. The incubation period from the time of virus

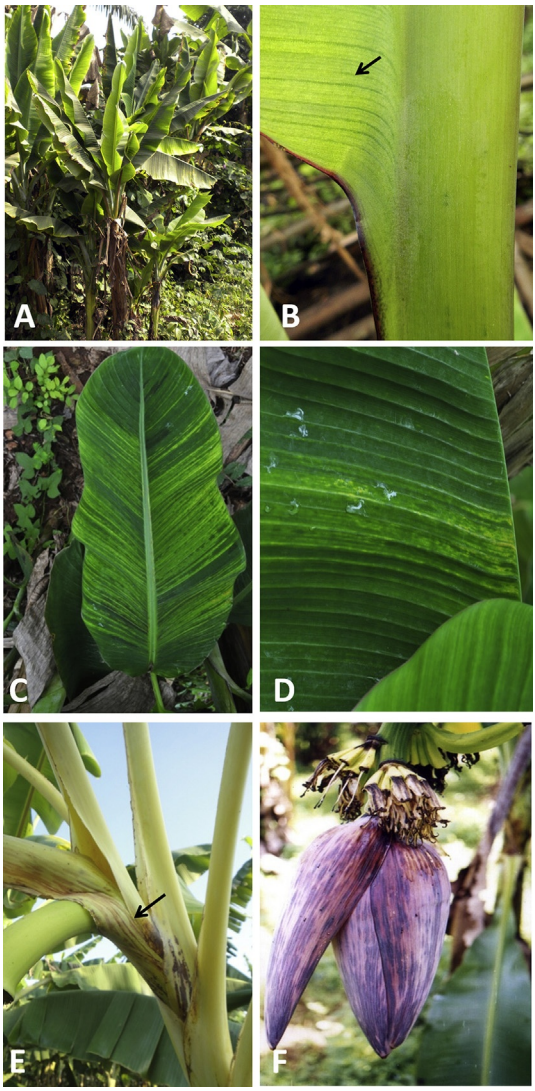

Figure 2 Severe bunching symptoms (A) and green streaks (B) symptoms in BBTV infected plant; yellow streak symptoms on leaves of the BSV infected plant (B and C); and BBrMV symptoms on petioles (D) and bracts (E).

inoculation to symptom expression varies between 19 and 125 days, depending on the stage of infection, cultivar, and weather (Allen, 1978; Hooks, Wright, Kabasawa, Manandhar, & Almeida, 2008). The shortest time for the diagnosis of BBTV using polymerase chain reaction (PCR) was 15 days after infection (Hooks et al., 2008). However, suckers emerging from the infected stools show symptoms from the time of emergence.

BBTD outbreaks in various countries have caused severe reductions in productivity. The first epidemic of BBTD recorded in Cavendish banana in Fiji reduced production from 788,000 bunches to 114,000 bunches at the end of the nineteenth century (Magee, 1953). A severe outbreak of BBTV in Australia in the 1920s destroyed approximately 90% of the crops in Queensland and New South Wales (Fist, 1970). One of the most significant BBTD epidemics was in Tamil Nadu, India, growing dessert banana cv. Virupakshi (Pome group, AAB), that reduced the production area from 18,000 to 2000 ha (Kesavamoorthy, 1980). The first BBTD epidemic in Sindh province in Pakistan reduced the production area from 60,000 to 26,000 ha (Khalid, Soomro, & Stover, 1993). New BBTD outbreaks 2007–2010 in Kodur, Andhra Pradesh, and Jalgaon, Maharashtra, India, caused an annual loss of production worth US$50 million (Selvarajan & Balasubramanian, 2014).

In Africa, BBTD is observed most commonly at elevations below 1300 m.a.s.l. (Kumar et al., 2011; Walangululu et al., 2010) and sparsely above 1700 m.a.s.l. in the hills in eastern DRC (Niyongere, Losenge, et al., 2011). The period of disease incubation is longer at higher altitudes (84 days at 2090 m.a.s.l.) compared with 21 days at 780 m.a.s.l. (Niyongere et al., 2013). Records of losses from BBTD outbreaks in Africa are lacking. In Malawi, a disease outbreak was first recorded in the mid-1990s; the estimated reduction in production was 50–95% at farm level and 30–50% at community level (Soko et al., 2009). Complete elimination of production was observed in Nkhatabay and Nkhotakota districts in the central region along Lake Malawi (Soko et al., 2009). BBTD has also been reported as a serious problem in Burundi, DRC, Gabon, and Rwanda and recently in Bénin, Nigeria, Congo Brazzaville, and the south of Cameroon. Although accurate estimates were lacking, a yield loss of 50–100% in severely infected plants of highly susceptible cultivars was reported from affected regions (Adegbola, Ayodeji, Awosusi, Atiri, & Kumar, 2013; Lokossou et al., 2012; Tushmereirwe & Bagabe, 1999). In general, mean incidence of BBTD in the affected regions across SSA varies from 5% to >70% and is known to result in variable production losses (Fouré & Manser, 1982; Kumar et al., 2011; Niyongere et al., 2013). Farmers in severely affected regions often abandon cultivation of the crop (Soko et al., 2009).

2.1.3 Transmission

BBTV is primarily disseminated through vegetative propagules, including the suckers, corms, and tissue-cultured plants (Drew, Moisander, &

Smith, 1989). The virus is vectored by a banana aphid, *P. nigronervosa* Coquerel, in which it is persistent and transmitted in a circulative manner (Anhalt & Almeida, 2008; Selvarajan, Balasubramanian, & Sathiamoorthy, 2006). Recently, another closely related species, *P. caladii* van der Goot, has been shown to transmit BBTV under experimental inoculation conditions but at a lower level of efficiency than *P. nigronervosa* (Watanabe, Greenwell, & Bressan, 2013).

Much of the attention on the transmission of BBTV by insects is focused on the banana aphid which is known to be highly specific to *Musa* species and present worldwide (Robson, Wright, & Almeida, 2006). Besides banana, host plants of *P. nigronervosa* include other members of the family *Musaceae*. On banana, aphids are frequently observed near the base of plants, followed by the newest unfurled leaf at the top (Robson et al., 2006). Studies in SSA indicated their distribution and abundance on *Musa* spp. are genotype dependent (Hanna et al., 2013). Aphids have a high rate of population growth at 25 °C compared with 20 or 30 °C (Robson, Wright, & Almedia, 2007). Allen (1987) estimated that the vector can spread virus from a primary source to a mean distance of 15.2 m in Australia.

In transmission experiments, the aphid was shown to transmit BBTV after the 4 h (minimum period) for acquisition access and 15 min of inoculation access (Hu, Wang, Sether, Xie, & Leonhardt, 1996). Further work by Anhalt and Almeida (2008) demonstrated a more efficient transmission of BBTV by adult aphids than by the third instar nymphs. Adults transmitted the virus more efficiently at 25 and 30 °C than at 20 °C. The minimum period for acquisition and inoculation for efficient transmission was in the range of 18–24 h. BBTV transmission by *P. nigronervosa* requires a latent period ranging from 20 to 28 h.

2.1.4 Geographic distribution and host range

BBTV is now known to occur in 36 countries; 14 are in Africa (Blomme et al., 2013; Kumar et al., 2011) and 22 in Asia and Oceania (Diekmann & Putter, 1996) (Fig. 1). BBTV recorded in Hawaii (Conant, 1992) was the only report of its occurrence in the New World. In Africa, occurrence of BBTD was first recorded from Egypt in 1901 (Fahmy, 1927), where there was an economically damaging spread of the disease in 1953 (Dale, 1987). BBTD was first discovered in SSA in the 1950s from DRC (Wardlaw, 1961) and later in Gabon, Burundi, Central African Republic, Equatorial Guinea, Rwanda, Malawi (Kenyon, Brown, & Khonje, 1997), Zambia (Gondwe, Mwenebanda, Natha, & Mutale,

2007), Angola (Kumar et al., 2008; Pillay, Blomme, Rodrigues, & Ferreira, 2005), and Cameroon (Oben et al., 2009). The first outbreak of BBTD in West Africa was recorded in the south-eastern part of the Republic of Benin (Lokossou et al., 2012) and spread from there into Nigeria (Adegbola et al., 2013). In Mozambique, the disease was seen in a banana nursery block in western Mozambique (Gondwe et al., 2007), but diseased plants were destroyed and new infections were not reported. An outbreak of BBTD was reported in Eritrea in 1964 (Saverio, 1964), but the current status is not known.

BBTV is known to infect natural and synthetic hybrids of *Musa paradisiaca*, abaca (*M. textilis*) (Manila hemp) (Sharman, Thomas, Skabo, & Holton, 2008) and *Ensete ventricosum* (Selvarajan & Balasubramanian, 2013). Searches for BBTV were negative in plants belonging to the species *Alpinia*, *Heliconia*, *Canna*, and *Strelitzia*, often found growing in the *Musa* production zones (Geering & Thomas, 1997). One report on BBTV detection in *Colocasia esculenta* in India (Ram & Summanwar, 1984) was not proved unequivocally (Geering & Thomas, 1997; Hu et al., 1996).

2.1.5 BBTV diversity

Various BBTV isolates characterized so far around the world have >85% homology (Banerjee et al., 2014). Although ABTV is also known to cause symptoms similar to those of BBTV in *Musa* spp., it is less prevalent and recognized so far only in the Philippines and Malaysia, mainly infecting abaca (Sharman et al., 2008). In general, the genetic diversity of BBTV isolates within the countries is very low [see studies in India (Selvarajan et al., 2010; Vishnoi, Raj, & Prasad, 2009), Pakistan (Amin, Qazi, Mansoor, Ilyas, & Briddon, 2008), Africa (Adegbola et al., 2013; Kumar et al., 2011), and Oceania (Stainton et al., 2012)]. However, in India, relatively greater diversity for BBTV was observed in the north-eastern region (Banerjee et al., 2014), including the identification of a new Babuvirus—*Cardamom bushy dwarf virus* (CBDV)—in cardamom (Mandal, Shilpi, Barman, Mandal, & Varma, 2013) (Fig. 3).

Based on the phylogenetic relationships among the DNA-R component sequences, various BBTV isolates were grouped into two different lineages: (i) the Pacific-Indian Oceans (PIO) group (formerly South Pacific group) comprising isolates in Africa, Australia, Hawaii, south Asia, Myanmar, and Tonga; and (ii) the South-East Asian (SEA) group (formerly Asian group) comprising isolates from China, Indonesia, Japan, the Philippines, Taiwan, and Vietnam (Banerjee et al., 2014; Karan, Harding, & Dale,

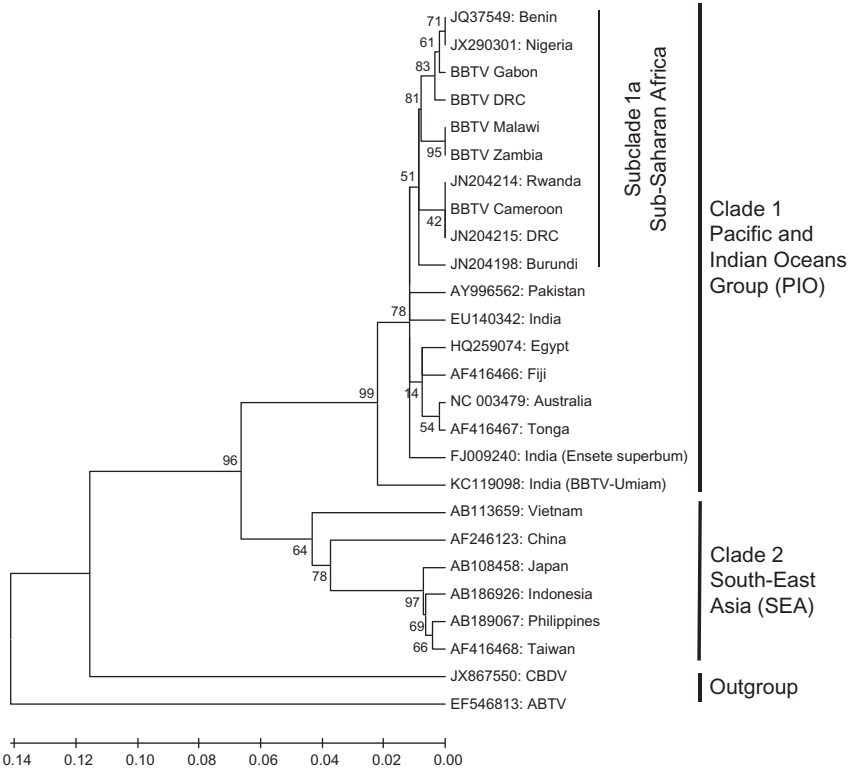

Figure 3 The evolutionary history of BBTV inferred using Neighbor-Joining method based on the ClustalW alignment of the 240 base pairs of Banana bunchy top virus (BBTV) DNA-R master replication-protein of various BBTV isolates. Country of origin of the virus isolates and corresponding NCBI GenBank accession numbers are listed. Bootstrap values (1000 replications) are shown as percentages at the branch points. *Abaca bunchy top virus* (ABTV) and Cardamom bushy dwarf virus (CBDV) are included as outgroup species using the Neighbor-Joining method. Phylogenetic analyses were conducted in MEGA4 (Tamura, Dudley, Nei, & Kumar, 2007).

1994; Stainton et al., 2012; Yu et al., 2012) (Fig. 3). Isolates from each country from these two groups are clustering together, indicating geographic separation. However, a BBTV isolate from Taiwan that has affinities with the PIO group is an exception (GenBank Accession no. EF095164).

Viral genome-based studies are also providing clues to the origin and pattern of BBTV spread. For instance, the DNA-R based sequence analysis of BBTV isolates from SSA, clustered together forming a unique sub-lineage within the PIO group, suggests a common origin for the isolates in SSA (Kumar et al., 2011) (Fig. 3). Historical data and farmers' knowledge of

disease occurrence suggest the first occurrence was near Yangambi in the Kisangani region, DRC (Wardlaw, 1961). Subsequent reports were from regions and countries adjoining DRC (Central African Republic, Congo Brazzaville, Gabon, Equatorial Guinea, Rwanda, Burundi, northern Angola, and northern Zambia) indicating a strong potential for virus dispersal through infected planting material along with human movement (Blomme et al., 2013; Kumar et al., 2011). The recent spread of BBTV into Bénin and Nigeria appears to be through the introduction of infected planting material from the Central African region (Adegbola et al., 2013; Lokossou et al., 2012). However, the isolate from Egypt, first reported in 1901, was aligned more closely with isolates from Australia and Fiji than with those from SSA (Fig. 3). These accounts support the possibility that BBTV was introduced into Africa in two separate events, one in North Africa in Egypt and the second in SSA, which contributed to pandemic there (Dale, 1987; Fouré & Manser, 1982; Kumar et al., 2011; Wardlaw, 1961) The pattern of spread in Hawaii is similar to that in SSA. Almedia, Bennett, Anhalt, Tsai, and Grady (2009), using molecular data, suggested that the inter-island spread of BBTV in Hawaii was driven by the human displacement of infected planting material. All this evidence underscores the need for appropriate containment procedures to prevent spread through planting material.

2.1.6 BBTV diagnostics

First detection methods for BBTV were based on enzyme-linked immunosorbent assay (ELISA) using monoclonal and polyclonal antibodies (Thomas & Dietzgen, 1991; Wu & Su, 1990b), and are available commercially (www.agdia.com). Various formats of ELISA (triple antibody sandwich ELISA, plate-trapped antigen ELISA, and double antibody sandwich ELISA) have been established for the reliable detection of the virus in field-grown plants, tissue culture plants, and aphids (Geering & Thomas, 1996; Selvarajan, Balasubramanian, Dayakar, Sathiamoorthy, & Ahlawat, 2010; Thomas & Dietzgen, 1991; Wu & Su, 1990b). Virus in the inoculated plants can be detected after 12–25 days depending on the genotype and stage of infection (Hooks et al., 2008) in any part of the infected plant; however, the most sensitive detection is in samples from the mid-rib portion of the youngest leaf. Methods based on nucleic acid spot hybridization (NASH) using DNA probes have been applied for the sensitive detection of BBTV (Hafner, Harding, & Dale, 1997; Harding et al., 1991; Selvarajan & Balasubramanian, 2008; Xie & Hu, 1995).

Methods based on the PCR have become an important technique for virus detection in plants and vectors, because of their superior sensitivity and flexibility (Galal, 2007; Hu et al., 1996; Thiribhuvanamala, Doraiswamy, & Ganapathy, 2005; Xie & Hu, 1995). DNA primers have been described for the amplification of BBTV, components 1–6, and virus-associated satellite DNAs. Differential primers to distinguish PIO and SEA isolates have also designed (Burns et al., 1995; Mansoor et al., 2005; Sharman, Thomas, & Dietzgen, 2000; Stainton et al., 2012). A simple virus release protocol from tissues without homogenization has been used in PCR as a rapid method for virus detection (Thomson & Dietzgen, 1995). Real-time PCR assays with TaqMan™ probes have been developed the quantitative detection of viral DNA segments in both plants and aphid tissues (Bressan & Watanabe, 2011; Chen & Hu, 2013). Recently, isothermal DNA amplification methods, such as Loop-mediated Isothermal Amplification (LAMP) (Peng, Fan, & Huang, 2012) and Rolling Circle Amplification (RCA) have been developed (Stainton et al., 2012). LAMP products can be detected using either conventional agarose gel electrophoresis or by visual observation of turbidity/color changes (Peng, Zhang, et al., 2012). The latter option is convenient for quick diagnosis in the field or in areas where molecular laboratory facilities are not available. RCA is gaining popularity as research tool for amplification of full-length BBTV DNA segments (Stainton et al., 2012). Recombinase Polymerase Amplification is another isothermal technique being developed for the detection of BBTV (M. Amato, personal communication). This method can be performed at temperatures between 37–42 °C, and is a fast emerging, simple DNA amplification technique for rapid and sensitive detection outside a laboratory (Piepenburg, Williams, Stemple, & Armes, 2006).

2.1.7 Options for BBTV control

2.1.7.1 Integrated disease control by exclusion, eradication, and use of virus-free plants

Banana cultivars fully resistant to BBTV are not available. However, some with the B genome (AAB and ABB) are tolerant or express symptoms more slowly than those with the A genome (AA and AAA), such as the Cavendish subgroup (Espino, Magnaye, Johns, & Juanillo, 1993; Jose, 1981; Ngatat et al., 2013). Various *Musa* clones vary in their degree of susceptibility, even among cultivars with only an A genome composition (e.g., Gros Michel) (Hooks, Manandhar, Perez, Wang, & Almeida, 2009; Hooks et al., 2008, Magee, 1948). Tolerant clones have been utilized in the BBTD endemic

areas, such as in Fiji (Magee, 1948), to recovery banana production. Current practices of BBTV management in many countries are based on the integration of several approaches, such as the eradication of infected plants by regular roguing, use of virus-free plants, and exclusion (quarantine) measures to prevent disease spread into other regions (Bouhida & Lockhart, 1990).

The availability of virus-free stocks is one of the major limitations in affected areas. *In vitro* methods have been established to generate virus-free planting material through meristem-tip culture combined with heat therapy (Lassois, Lepoivre, Swennen, van den Houwe, & Panis, 2013; Thomas, Smith, Kessling, & Hamill, 1995). These virus-free plants are then used as mother stocks for the mass propagation of virus-free planting material (Su, Hwang, Lee, & Chao, 2007). This approach, backed with certification systems, is now accepted for disease control in many countries in Asia, also in Australia and Hawaii. In some countries, such as India, recurring outbreaks in farmers' fields led to the formulation and strict enforcement of certification systems and commercial production units have been accredited to produce certified tissue cultured plants. More than 22 million TC plants were certified in 2013–2014 alone (R. Selvarajan, personal communication). In Australia, only pathogen-free stocks generated by the Queensland Banana Accredited Nursery (QBAN) are allowed to be used as foundation stock in the TC industry (QPPR, 2002). Presently, virus-free TC plants are widely used to manage BBTV in Taiwan, the Philippines, and India (Molina et al., 2009; Selvarajan, Balasubramanian, Sheeba, Raj Mohan, & Mustaffa, 2011; Su et al., 2007). However, in many SSA countries there are no guidelines or certification schemes for producing high quality planting material, thus increasing the risk of pathogen spread from the unregulated movement of uncertified planting material from one area to another. A very recent study in south-western DRC by Mukwa et al. (2014) attributed the high prevalence of BBTV in the region to the multiple introductions of infected planting materials in Bas Congo province, 1990–2002.

New plantations established in an endemic region are exposed to a high risk of infection. Regular monitoring has been recommended to identify and rogue infected plants to reduce virus spread as soon as symptoms appear (Allen, 1987; Hooks et al., 2008; Magnaye, 1994; Smith, Holt, Kenyon, & Foot, 1998). Allen (1987) also recommended uprooting apparently symptomless plants within 5 m radius of symptomatic plants as a precautionary principle. This however has been shown to result in the elimination of a considerable number of uninfected plants for every infected plant uprooted.

Injecting herbicides, such as fernoxone or 2,4-D, together with systemic insecticide sprays has been recommended to kill the infected plants and prevent the spread of aphids from the treated plants (Regupathy, Subramanian, & Naganathan, 1983; Robson et al., 2007). These methods are labor intensive, costly for many farmers, and of questionable effectiveness in smallholder farming systems where all farmers may not adopt common practices.

So far, intensive eradication of BBTV has been implemented only in Australia. These efforts have resulted in marked reductions in the prevalence of the virus, although outright eradication was not achieved (Allen, 1987; Cook et al., 2012). Using Monte Carlo simulation, Cook et al. (2012) estimated that the implementation of exclusion measures in Australia prevents annual losses of Aus$15.9–27.0 million for the banana industry. To have a similar program in developing countries where the crop is grown dominantly by smallholder farmers could be highly challenging. Recently, under CGIAR Roots Tubers and Banana Program, IITA, Bioversity International, CIRAD, and national programs in SSA partnered to implement a new initiative to contain BBTD (www.rtb.cgiar.org). This pilot phase, from 2014 to 2016, implemented in Bénin, Burundi, Cameroon, Congo Brazzaville, DRC, Malawi, and Nigeria, aims to pilot eradication of infected plants and recover production using healthy planting material in selected sites.

2.1.7.2 Host resistance

Use of disease resistant cultivars offers the most convenient and effective solution for the control of plant diseases. A diverse range of banana and plantain cultivars have been evaluated in Asia and Africa (Espino et al., 1993; Hooks, Manandhar, et al., 2009; Hooks et al., 2008; Jose, 1981, Magee, 1948; Niyongere, Ateka, Losenge, Lepoint, & Blomme, 2011). All these studies highlighted the lack of high levels of resistance to BBTD infection but tolerance was found, typified by a low or slow rate of infection. In field evaluation trials from 2004 to 2006 in Malawi, all 22 cultivars, including banana, plantain, and hybrids, were found to be susceptible to BBTV although with differences in symptom severity (Mwenebanda, Mwamlima, Msosa, & Banda, 2007). In an on-going trial in a BBTV endemic location in southern Cameroon, 16 *Musa* genotypes expressed varied symptoms (Ngatat et al., 2013). Williams (AAA) and the hybrid plantain PITA 23 (AAB) were the most susceptible with >90% infection. Local plantain landraces (AAB), several hybrid plantain (AAB and AAAB), a cooking banana (AAB), and Grande Nain (AAA) were moderately susceptible with a range of infection from about 30–60% infection. Gros Michel (AAA) and

Pisang Awak (ABB) were found to be least susceptible with infection less than 20%.

To overcome the current limitations of low resistance to BBTV in the *Musa* germplasm and breeding challenges for edible *Musa* spp., transgenic approaches based on the pathogen-derived resistance (PDR) strategies are being explored. Strategies using BBTV DNA-R gene or satellite DNA (DNA-S4) resulted partial resistance to BBTV (Tsao, 2008). More recent efforts using RNA interference (RNAi) have resulted in the development of clones with near immunity to infection (Borth et al., 2011; Elayabalan, Selvarajan, & Kumar, 2013). Shekhawat, Ganapathi, and Hadapad (2012) used intron-hairpin-RNA transcripts corresponding to the viral master replication initiation protein (Rep) to generate the BBTV-resistant transgenic banana cv. Rasthali. Transgenic plants challenged and inoculated with viruliferous aphids tested negative to infection 6 months after inoculation. Although field evaluation of these transgenic clones is still due, this approach has shown great potential to establish resistant clones and might prove useful to induce PDR in several popular *Musa* cultivars. Recently, IITA and Queensland University of Technology (QUT) have initiated an RNAi-based transgenic program to develop banana and plantain resistant to BBTV and aphids for Africa (L. Tripathi, personal communication).

Mutation breeding using gamma-irradiation has also been explored to induce genotyping and phenotyping variation for BBTV resistance. In the Philippines, mutation breeding through the gamma-irradiation of shoot-tip explants of cv. Lakatan (AAA) resulted in lines with resistance against BBTV (Damasco et al., 2006; Dizon et al., 2012). Of the 7366 regenerated plants screened, 29 lines were found to be highly resistant to the virus. Resistance in these lines was found to be due to the aphids' non-preference to colonize mutant banana lines and reproduce. Virus multiplication was reduced as indicated by low titer on mutant lines (Abustan, 2012).

Although significant progress has been achieved in the past decade to improve the levels of resistance in popular cultivars through transgenic approaches, none of these clones are available for commercial cultivation.

2.1.7.3 Vector control

Options for control of the banana aphid have so far received little attention. Pesticides for banana aphid control have been used mainly as a pre-emptive measure to prevent their spread from withering plants during eradication programs (Hooks, Fukuda, et al., 2009). It is unlikely that vector control

using pesticides will gain popularity because of prohibitive costs and concerns with residual effects on health and environment. Biological control is so far not available as no natural biocontrol agents have been identified (R. Hanna et al., unpublished).

2.2. Banana streak disease

2.2.1 Disease discovery and biology

BSV causes chlorotic streak disease and is known to be the most widely distributed virus infecting banana and plantain around the world. The disease was first observed in the Nieky Valley on the Ivory Coast in 1958 (Lockhart & Jones, 2000) and later, in 1964, severe BSV chlorosis disease was reported in the Gros Michel triploid *M. acuminata* (AAA) banana cultivar. Lockhart (1986) purified bacilliform particles from field-grown Dwarf Cavendish banana (AAA) in southern Morocco, confirming the viral etiology of the disease. The data currently available on the disease show a complex of distinct BSVs, each causing the same disease.

BSVs are pararetroviruses belonging to the genus *Badnavirus*, family *Caulimoviridae*. The virus particles are bacilliform-shaped (120–150 × 30 nm), double-stranded noncovalently closed circular DNA (dsDNA) genome approximately 7.2–7.8 kb long that uses a virus-encoded reverse transcriptase (RT) to replicate. Harper and Hull (1998) were the first to describe the structure of the virus genome and named it *Banana streak Obino l'Ewai virus* (BSOLV). The virus genome has three consecutive ORFs on one strand (King et al., 2012). ORF1 and ORF2 potentially encode two small proteins of unknown function of 20.8 and 14.5 kDa. ORF3 is a large polyprotein of 220 kDa encoding at least four proteins, encompassing a putative cell-to-cell movement protein, a coat protein (analogous to retroviral GAG), an aspartic protease, and a viral replicase consisting of RT and RNase H domains (Harper & Hull, 1998; King et al., 2012). This polyprotein is cleaved into functional units by the aspartic protease once it has been fully translated. In contrast to retroviruses, BSV does not encode integrase, neither does it require integration into the host genome to replicate.

Two infectious forms of BSV exist: (i) the episomal form resulting from cells/plant infection following transmission by mealybugs and (ii) endogenous forms which are endogenous viral sequences of BSV (eBSV) integrated within the banana B genome (*M. balbisiana*). Physical stresses have been reported to induce *de novo* viral particles (episomal form) from eBSV (Cote et al., 2010; Lheureux, Carreel, Jenny, Lockhart, & Iskra-Caruana, 2003), possibly through intra-strand homologous recombination (HR)

(Chabannes & Iskra-Caruana, 2013; Iskra-Caruana, Baurens, Gayral, & Chabannes, 2010). Both episomal virus and infectious particles from eBSV give rise to systemic plant infection (Harper, Ganesh, Thottappilly, & Hull, 1999, Harper, Hart, Moult, & Hull, 2004; Iskra-Caruana et al., 2010). BSV particles from both origins can be transmitted by mealybugs (Dahal et al., 2000; Kubiriba, Legg, Tushemereirwe, & Adipala, 2001; Lockhart & Autrey, 1988)

2.2.2 Symptoms

BSVs infect different species of *Musa* and the natural and synthetic hybrids. Most isolates produce discontinuous yellow dots/streaks that turn necrotic on the leaves, and also pseudo-stem splitting (Fig. 2). Generally, symptoms are erratically distributed on the plant and not shown on all leaves. Some isolates immediately produce stunting with severe necrotic streaks resulting in cigar leaf necrosis and leading quickly to the death of the plant (Thangavelu, Selvarajan, & Singh, 2000). Symptomatic and symptomless stages alternate in infected plants but virus can be detected at all stages (Harper et al., 2002; Lassoudière, 1974; Lockhart & Jones, 2000).

Lassoudière (1974) recorded yield losses ranging up to 90% on Poyo (AAA, Cavendish subgroup) producing few if any exportable bunches. However, Daniells, Geering, Bryde, and Thomas (2001) observed only a mild effect (about 10% loss) on Cavendish subgroup cv. Williams triploid *M. acuminata* (AAA) under good growth conditions for banana with only a delay of bunch emergence and maturation.

2.2.3 Transmission and geographic distribution

BSV is not mechanically transmitted and field spread occurs by semi-persistent mealybug-mediated transmission and by the use of infected planting material, such as suckers. Field observations suggest that virus spread is slow (Daniells et al., 2001) with no difference in isolate transmission observed between the two main vector species *Planococcus citri* and *Pseudococcus* spp. (Dahal et al., 2000; Kubiriba et al., 2001; Matile-Ferrero & Williams, 1995). Other species reported to transmit BSV are *Dysmicoccus* spp. in West Africa and South America, *Planococcus musa* in Nigeria, *Ferrisia virgata* (striped mealybug) in India (Selvarajan et al., 2006), *D. brevipes* and *P. ficus* (Meyer, Kasdorf, Nel, & Pietersen, 2008) and *Paracoccus burnerae* (Muturi, Wachira, Karanja, Wambulwa, & Macharia, 2013) in South Africa. Transmission assays using *P. burnerae* demonstrated the inability of the vector to acquire and transmit the virus during hot conditions (24–30 °C).

However, under cool conditions (9–20 °C), a minimum of 6 h of feeding time was necessary for *P. burnerae* instars to become viruliferous (Meyer et al., 2008).

BSV is now reported to occur in over 43 countries of Africa, Asia, Australia, Europe, Oceania, and tropical America (Diekmann & Putter, 1996) (Fig. 1). However, the epidemiology of BSV remains surprisingly unclear and the role of eBSV still needs to be clarified. Iskra-Caruana, Chabannes, Duroy, and Muller (2014) proposed an evolutionary history based on both field studies and molecular epidemiology to explain the current situation, providing insights into the ecology and evolution of BSVs and *Musa* sp.

2.2.4 Virus diversity

The genus *Badnavirus* is both the most complex and the most diversified genus within the family *Caulimoviridae*, with at least three major clades (Harper et al., 2004, 2005; King et al., 2012). A final phylogeny of BSVs has been established to clarify whether partial sequences distributed over the three main clades of the genus *Badnavirus* correspond to episomal viruses with or without an endogenous counterpart (Gayral & Iskra-Caruana, 2009; Iskra-Caruana, Chabannes, Duroy, et al., 2014; Iskra-Caruana, Duroy, Chabannes, & Muller, 2014) (Fig. 4). Clades 1 and 3 are dedicated to BSV and Clade 2 gathers all *Musa* endogenous badnavirus sequences, with no episomal counterpart reported so far Chabannes et al., in preparation. Clade 1 also groups the four BSV species having an eBSV counterpart in the B genome (BSOLV, BSGFV, BSIMV, and BSMYV); Clade 3 groups only BSV species of Uganda (Fig. 4).

2.2.5 Diagnostics

Detection of BSV was first achieved using serological approaches although detection is complex due to the occurrence of a wide degree of diversity among virus isolates, some of which are serologically unrelated (Lockhart & Olszewski, 1993; Ndowora, 1998). For this purpose, a polyvalent polyclonal antiserum against BSV and *Sugarcane bacilliform virus* spp. has been developed (B.E.L. Lockhart, unpublished). It is capable of detecting most known BSV isolates but fails to detect some endemic BSV species (BSUIV, BSUJV, BSULV, BSUMV) from Uganda belonging to Clade 3 (Chabannes et al., unpublished). Meyer (2005) developed two other polyvalent BSV polyclonal antisera from 20 BSV isolates present in South Africa; these gave no background with plants but one failed to detect BSMYV

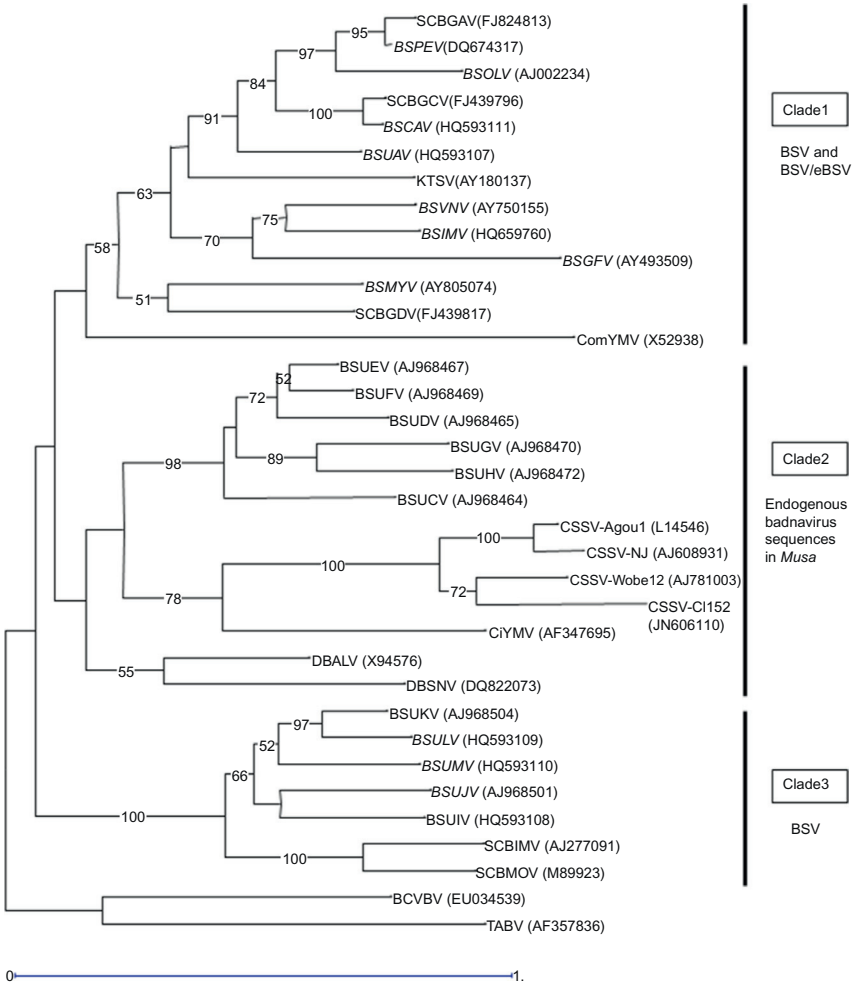

Figure 4 Maximum likelihood phylogeny of badnavirus sequences based on alignment of a 540-bp fragment of the RT/RNase H viral region. Bootstrap values of 500 replicates are given when >50%. *Taro bacilliform virus* (TABV) and *Bougainvillea spectabilis chlorotic vein-banding virus* (BCVBV) are given as outgroups. Viral sequences isolated from *Musa* are in bold. BSV species where a full-length sequence is available are in italic. The scale bar shows the number of substitutions per base. The GenBank accession numbers of sequences are given in parenthesis. *Cacao swollen shoot virus*-CSSV-Agou1, *Commelina yellow mottle virus*-ComYMV, *Citrus yellow mosaic virus*-CiYMV, *Dioscorea bacilliform AL virus*-DBALV, *Dioscorea bacilliform SN virus*-DBSNV, *Kalanchoe top-spotting virus*-KTSV, *Sugarcane bacilliform MO virus*-SCBMOV, *Sugarcane bacilliform IM virus*-SCBIMV, *Sugarcane bacilliform Guadeloupe A virus*-SCBGAV, *Sugarcane bacilliform Guadeloupe C virus*-SCBGCV, and *Sugarcane bacilliform Guadeloupe D virus*-SCBGDV. *Adapted from Iskra-Caruana, Duroy, (2014) and Iskra-Caruana, Chabannes, et al. (2014).*

(M.L. Iskra-Caruana, unpublished). Agindotan, Thottappilly, Uwaifo, and Winter (2003) reported high titered monoclonal antibodies for the detection of all isolates of BSV. Chen, Rao, Ruan, Liu, and Li (2013) reported the production of antibodies to the recombinant coat protein domain of BSV-Guangdon isolate which was effective at 1:600 to 1:6400 (v/v) dilutions in detecting BSV in infected plants. Serological detection of BSV can be achieved by different techniques, such as immunosorbent electron microscopy (ISEM) (Ndowora, 1998); DAS-ELISA (Meyer, 2005; Thottappilly, Dahal, & Lockhart, 1998) or Immuno-capture PCR (IC-PCR) (Harper, Osuji, Heslop-Harrison, & Hull, 1999; Le Provost, Iskra-Caruana, Acina, & Teycheney, 2006). Detailed and updated protocols for these three techniques can be found in the *Musa*Net Technical Guidelines (2014) for the safe movement of *Musa* germplasm. IC-PCR is more sensitive than immuno-electron microscopy (IEM) for detecting typical BSV; IEM sensitivity was proved to be similar to TAS-ELISA by sap dilution end point analyses (Agindotan et al., 2006). ISEM requires a partially purified plant extract and an electron microscope but is sensitive because BSV particles can be detected even in asymptomatic leaf tissue (B.E.L. Lockhart, unpublished). ELISA kits are now commercially available for BSV (www.sediag.com). Although both ELISA and ISEM techniques can detect most BSVs, species identification is not possible.

Diagnosis by PCR does not distinguish between infections (episomal) and eBSV. In addition, all *Musa* spp. carry partial or "dead" (truncated or mutated) integrated badnaviral sequences in their genomes that are incapable of generating infectious particles. Thus, PCR tests on any total genomic DNA extracted from a plant using moderate degenerated BSV primers may result in BSV-positive reactions. IC-PCR allows the detection of episomal particles after the binding of BSV to the polyvalent polyclonal antiserum and also identifies viral species using specific BSVs primers. This molecular approach is now the most commonly used diagnostic test for BSV. However, the presence of eBSV in *Musa* genomes could still interfere with PCR-episomal detection and leads to false positive results. To overcome this problem, Le Provost et al. (2006) used *Musa* sequence tagged microsatellite site primers developed by Lagoda et al. (1998) to detect genomic DNA contamination. These primers were used in multiplex with specific BSV species primers. Alternatively, a DAase I treatment step can be performed after the immune-capture stage to remove any plant DNA contamination (Chabannes et al., in preparation).

More recently, two new molecular techniques have been proposed as an alternative for the detection of BSV particles: the LAMP assay (Peng, Fan, et al., 2012) and RCA (James, Geijskes, Dale, & Harding, 2010, 2011). Both methods have the advantage of amplifying the target DNA without a thermal cycling instrument. RT multiplex PCR has been applied for the detection of BSV to avoid detection of eBSV sequences (Liu et al., 2012; Selvarajan et al., 2011). However, exclusive detection of episomal BSV by this method is difficult because eBSV sequences are known to be transcribed resulting in RNA transcripts that can be detected by RT-PCR leading to wrong assumptions.

2.2.6 Control

The main control method to limit BSV infection is by the production and multiplication of healthy banana plants. As mealybugs are slow moving; the spread of the disease is therefore easy to restrict by removing infected plants. Mass propagation is achieved either by *in vitro* plantlet multiplication, as used mainly for dessert banana and commercial plantations, or by the use of suckers. Vegetative multiplication from infected plants can significantly increase BSV incidence in the field. For instance, high BSV incidence in Uganda seems to be the result of the mass propagation of plantlets from symptomless BSV-infected plants. However, in vitro multiplication remains the most appropriate way for genotypes free of eBSVs to supply large quantities of safe *Musa* planting material without pathogens, such as bacteria, fungi, and nematodes. Only a few reports have been published regarding therapy procedures to eliminate BSV from infected plant tissues and the regeneration of BSV-free planting material. Helliot et al. (2002) developed a cryopreservation followed by apical meristem culture that significantly reduces virus titers. The same authors also demonstrated the use of antiviral, compounds adefovir, tenofovir, and 9-(2-phosphonomethoxyethyl)-2,6-diaminopurine (PMEDAP), to eradicate episomal forms (Helliot et al., 2003).

Efforts to breed cultivars resistant to streak disease are limited. Many A-genome *Musa* cultivars were found to be susceptible to BSV infection, whereas several BB genome cultivars are known to be resistant. Interestingly, natural interspecific hybrids showed high tolerance to BSV expression from eBSV (M.-L. Iskra-Caruana, unpublished). Dahal et al. (2000) evaluated 36 genotypes with diverse genetic backgrounds, including 14 tetraploid plantain (TMPx) and banana (TMBx) hybrids developed by IITA, and hybrids from Fundacio´ n Hondureo‘ a de Investigacio´ n Agri‘cola

(FHIA), for their response to BSV expression in Nigeria. Some of the evaluated hybrids (e.g., TMPx 548-9, TMPx 2637-49, TMPx 7002-1, and FHIA 21) despite being infected gave near normal yields and were regarded as disease tolerant. Developing transgenic BSV resistance has so far not succeeded (Tripathi, 2003).

The main problem, encountered for over 20 years, concerns the presence of infectious eBSV for at least three BSV species (BSOLV, BSIMV, and BSGFV) in all *M. balbisiana* genomes (Chabannes et al., 2013; Duroy, 2012; Gayral et al., 2008, 2010) that are able to release infectious particles spontaneously in interspecific contexts (AB, AAB, or AAAB) when hybrids are exposed to stresses such as wounding, heat shock, or *in vitro* culture. Therefore, eBSV became the main constraint to controlling BSV and for safe exchange of *Musa* germplasm. Recently, three infectious eBSV species were characterized in the seedy diploid *M. balbisiana* genitor Pisang Klutuk Wulung (PKW) (Chabannes et al., 2013; Gayral et al., 2008). This information was used to develop molecular markers to distinguish both eBSV (haplotypes) and alleles (infectious vs. non-infectious for BSOLV and BSGFV) (Fig. 5). These eBSV diagnostic markers have been used already to genotype the Bioversity International *M. balbisiana* germplasm collection. They have also greatly helped CIRAD breeders to produce by self-pollination and doubled haploid lines, *M. balbisiana* progenitors devoid of infectious eBSOLV and eBSGFV by segregation of eBSOLV and eBSGFV alleles (Pichaut et al., 2013; Umber et al., in preparation). Introduction of these improved diploid *M. balbisiana* plants which harbor only noninfectious eBSVs to the *Musa* breeding program can abolish the risk linked to the presence of eBSVs. Very recently, using such markers, Noumbissié (2014) demonstrated the possibility of producing an eBSV-free (i.e., without infectious and non-infectious eBSV) triploid hybrid (AAB) using a conventional ($4\times/2\times$) breeding strategy. These molecular tools, which are free for use by the worldwide *Musa* community, should in the next few years help to control the dissemination of BSV through the distribution of newly created interspecific banana hybrids.

2.3. Banana bract mosaic

2.3.1 Distribution and biology

Banana bract mosaic disease, caused by the BBrMV, was first reported in 1979 in the Philippines (Magnaye & Espino, 1990; Rodoni, Ahlawat, Varma, Dale, & Harding, 1997). Occurrence of the virus was discovered

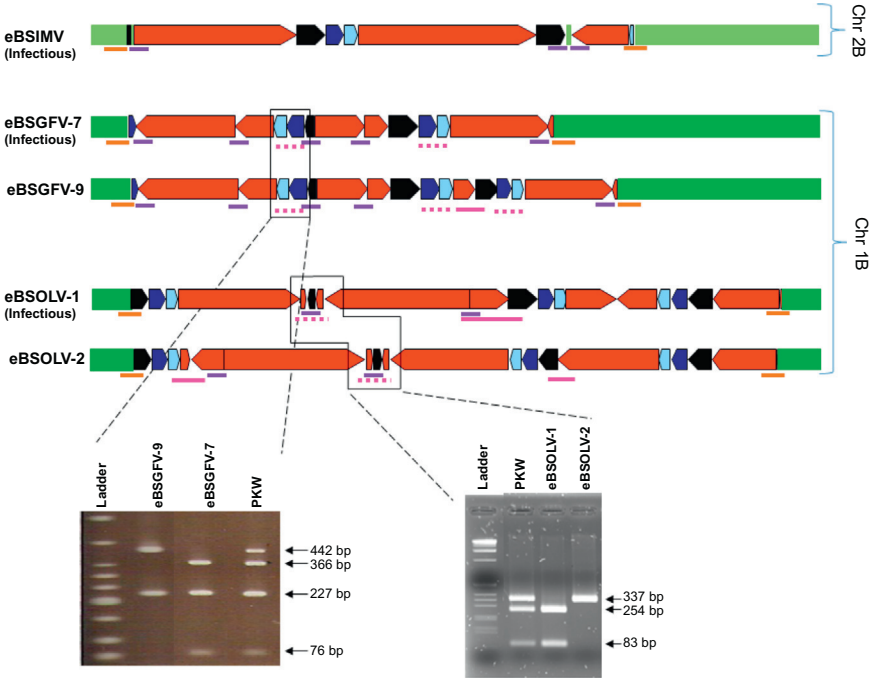

Figure 5 eBSV genotyping markers for eBSIMV, eBSGFV, and eBSOLV in Pisang Klutuk Wulung (PKW). Banana genomic sequences of chromosome 1 and 2 are in green. Dark blue, light blue, red, and black arrowed boxes represent ORF1, ORF2, ORF3, and the intergenic region (IG) of the BSV genome. Arrows indicate the orientation of the viral fragment. PCR fragments are indicated by color lines. Orange, purple, and pink, lines correspond to *Musa*-eBSV junctions, internal eBSV rearrangements, and eBSV allele-specific markers. Two kinds of markers have been developed to specifically discriminate alleles of each eBSV species: pink -full lines represent classical PCR markers whereas pink -dotted lines represent dCAPS markers. Agarose gel images below correspond to eBSGFV (left) and eBSOLV (right) allelic patterns obtained with dCAPS markers. PKW exhibits both alleles for each eBSV species.

in a few other countries in Asia and the South Pacific, including India, Samoa, Sri Lanka, Thailand, and Vietnam (Diekmann & Putter, 1996; Rodoni et al., 1997; Rodoni, Dale, & Harding, 1999). In Latin America, BBrMV occurrence was first reported in Colombia (Alarcon et al., 2006, cited in Quito-Avila et al., 2013). Recently, occurrence of this virus in a commercial farm was recorded in Ecuador (Quito-Avila et al., 2013). In Hawaii (USA), BBrMV was detected in ornamental ginger plants (*Alpinia purpurata*) but not in *Musa* (Wang, Sether, Melzer, Borth, & Hu, 2010).

BBrMV causes characteristic spindle-shaped, purplish streaks on bracts, pseudostems, midribs, peduncles, and even fruits (Rodoni et al., 1997; Selvarajan & Jeyabaskaran, 2006; Thomas, Geering, Gambley, Kessling, & White, 1997). Bunches from infected plants unusually contain a long or very short peduncle and in some cultivars, such as Nendran, the leaves appear as "traveler's palm" plant (Balakrishnan, Gokulapalan, & Paul, 1996). Necrotic streaks on fruits, leaves, pseudostems, and midribs have also been recorded (Selvarajan & Jeyabaskaran, 2006). Yield losses of between 30% and 70% have been recorded in India and the Philippines (Cherian, Menon, Suma, Nair, & Sudheesh, 2002; Magnaye & Espino, 1990; Thangavelu et al., 2000).

2.3.2 Host range and transmission

The main host of BBrMV is *Musa* spp., including abaca, widely grown in the Philippines (Sharman, Gambley, Oloteo, Abgona, & Thomas, 2000). Other natural hosts include small cardamom in India (Siljo, Bhat, Biju, & Venugopal, 2011) and flowering ginger, *A. purpurata*, a popular cut flower and tropical landscape plant in Hawaii (Wang et al., 2010). BBrMV is primarily transmitted through infected planting material. The virus is transmitted nonpersistently by several aphid species viz., *P. nigronervosa*, *Rhopalosiphum maidis*, *Aphis gossypii* (Magnaye & Espino, 1990; Munez, 1992), and *A. craccivora* (Selvarajan et al., 2006).

2.3.3 Virus diversity

BBrMV particles are flexuous and filamentous of ca. 750×11 nm with a single-stranded positive-sense RNA as its genome (Bateson & Dale, 1995; Thomas et al., 1997). The RNA genome is 9711 nucleotide long excluding the 3′-terminal poly A tail and contains a typical large ORF of 9378 nucleotides coding for a poly protein of 3125 amino acids, with 9 protease cleavage sites, potentially yielding 10 matured functional proteins that have motifs conserved among homologous proteins of other potyviruses (Balasubramanian & Selvarajan, 2012; Ha et al., 2008; Rodoni et al., 1997). The whole genome of BBrMV-TRY (India) and BBrMV-PHI (the Philippines) had 94% nucleotide sequence identity and 88–98% amino acid sequence identities (Balasubramanian & Selvarajan, 2012). The coat protein-encoding gene and 3′-untranslated region of BBrMV isolates from India, the Philippines, Samoa, and Vietnam, are 95.4–99% identical (Rodoni et al., 1999). The genetic diversity and recombination analysis in the CP gene of 49 isolates of BBrMV revealed a greater variation among

them and two of the isolates from Tamil Nadu were distinct with 18–21% divergence at nucleotide level and 12–20% divergence at amino acid level (Balasubramanian & Selvarajan, 2014). Eleven putative recombinants were also detected; whereas the partial gene sequences from Hawaii and Ecuador are 99% identical, compared with the corresponding fragment of a BBrMV isolate from the Philippines (Quito-Avila et al., 2013; Wang et al., 2010).

2.3.4 Diagnosis

A range of diagnostic methods based on ELISA (Espino, Exconde, Zipagan, & Espino, 1990, Espino, Exconde, Zipagan, Maroon, & Espino, 1989; Thomas et al., 1997), RT-PCR (Bateson & Dale, 1995; Ha et al., 2008; Kiranmai et al., 2005; Thomas et al., 1997, Exconde, Zipagan, Maroon, & Espino,), and IC-RT-PCR has been used for the detection of BBrMV singly (Iskra-Caruana, Galzi, & Laboureau, 2008) or in combination with other banana infecting viruses in a multiplex diagnostic assay (Liu et al., 2012; Selvarajan, Balasubramanian, Dayakar, Sathaimoorthy, & Ahlawat, 2004; Sharman, Thomas, et al., 2000).

2.3.5 Control

Sources of resistance have not been reported for BBrMV. Standard control measures include the removal and destruction of affected plants along with the rhizome and the use of virus-free sources as planting material. Application of fertilizer was reported to mitigate the symptoms of BBrMV in certain commercial banana cultivars (Selvarajan et al., 2009). Recently, abaca cvs. Tinawagan Pula and Tangongon with putative resistance to BBrMV were developed through *in vitro* mutagenesis by gamma-irradiation (Dizon et al., 2012). Less than 2% of over 3000 plants irradiated were found to be virus negative even 3 months after inoculation. These promising lines have yet to be field tested or evaluated for other agronomic traits. Nonetheless, this approach demonstrated the promise of mutagenesis in developing virus resistant cultivars.

3. MINOR VIRUS DISEASES

3.1. Abaca bunchy top

Abaca bunchy top is caused by ABTV, a distinct member in the genus *Babuvirus*, family *Nanoviridae* (Sharman et al., 2008). The virus has been recorded from Malaysia and the Philippines (Ocfemia, 1930). ABTV causes

vein clearing flecks and narrow, brittle leaves with chlorotic upturned margins in abaca; in Cavendish banana, symptoms are indistinguishable from those of BBTV (Diekmann & Putter, 1996; Su, Tsao, Wu, & Hung, 2003). The virus is transmitted through vegetative planting material and tissue cultured plants derived from infected sources. There are no reports on vector transmission. However, since the virus is a member of the genus *Babuvirus*, it is conceivable that the banana aphid, *P. nigronervosa*, may able to transmit ABTV in a persistent and circulative manner similar to BBTV. PCR assays have been established for ABTV detection (Sharman et al., 2008). Economic significance of this virus is not known. Since virus symptoms are similar to those of BBTV infection in banana, it may have a similar impact.

3.2. Abaca mosaic

Abaca mosaic is caused by SCMV-Ab, a distinct strain of SCMV (genus *Potyvirus*) (Eloja & Tinsley, 1963; Thomas et al., 1997). This virus has so far been recognized only in the Philippines. It induces spindle-shaped yellow chlorotic streaks on leaves, petioles, and midribs. The disease affects fiber yield and quality, and is a significant constraint to abaca production in the Philippines. Natural hosts recognized so far include *M. textilis*, *Maranta arundinacea*, and *Canna indica*. The virus is transmitted by vegetative propagation and tissue culture, also by aphids (mainly *R. maidis* and *A. gossypii*) in a nonpersistent manner (Diekmann & Putter, 1996). ELISA and RT-PCR assays have been established for virus detection (Gambley, Thomas, Magnaye, & Herradura, 2004). There are no specific recommendations for disease control, other than the use of clean planting material.

3.3. Banana mosaic

Banana mosaic, also known as infectious chlorosis, caused by CMV is one of the common viral diseases affecting banana and plantain worldwide. It causes variable symptoms from mild chlorosis to severe chlorotic streaks that are similar to the symptoms of BSV infection. Symptoms are known to fluctuate during the growing season depending on the temperature and rainfall. The virus has a wide host range, infecting over 900 species in almost every country with extensive agriculture. Most isolates of CMV from banana (CMV-B) have been identified to belong to subgroup I. Most common CMV strains do not produce severe symptoms or cause significant damage on banana. However, some severe strains cause severe necrosis in pseudostems (heart-rot) and even plant death (Bouhida & Lockhart, 1990).

The virus is transmitted through planting material and nonpersistently by several aphids, such as *A. gossypii*, *A. craccivora*, *R. maidis*, *R. prunifolium*, and *Myzus persicae* (Rao, 1980). CMV can be detected by several methods, such as mechanical inoculation to herbaceous plants, ELISA, NASH, RT-PCR methods, and electron microscopy (Hu, Li, Barry, Wang, & Jordan, 1995; Kiranmai, Sreenivasulu, & Nayudu, 1996). Kouassi, Wendy, Boonham, and Smith (2010) developed a quantitative real-time PCR (qRT-PCR) for screening banana planting material for CMV infection in Côte d'Ivoire. A RT-LAMP assay has also been established for CMV detection in banana (Peng, Shi, Xia, Huang, & Fan, 2012). Disease control is primarily through use of clean planting material. *In vitro* methods for the elimination of CMV from tissues are in common use (Helliot et al., 2002).

3.4. Banana mild mosaic

The disease is caused by BanMMV, an unassigned virus in the *Betaflexiviridae* (King et al., 2012). The virus has flexuous filamentous particles of about 580 nm in length, with a coat protein of ca. 26.8 kDa, and a single-stranded RNA genome (Gambley & Thomas, 2001). The virus often occurs in symptomless plants of *Musa* spp. and apparently has a worldwide distribution. It is often detected in mixed infection with BSV and BBrMV (Iskra-Caruana et al., 2008) and is reported to cause necrotic streaks in the presence of CMV infection in Guadeloupe (Iskra & Galzi, 1998). The virus spreads through the vegetative propagation of planting materials. Although a vector has not been identified, horizontal transmission through some unknown mechanism has been conjectured, based on the high heterogeneity of the viral genome and the temporal increase in virus incidence within a field (Teycheney, Laboureau, Iskra-Caruana, & Candresse, 2005). Polyclonal antisera to BanMMV have been developed which are used for virus detection by IC-RT-PCR and ISEM (Teycheney, Acina, Lockhart, & Candresse, 2007; Teycheney, Laboureau, et al., 2005). The virus is controlled mainly through the production and distribution of clean planting material. Meristem cultures and chemotherapy, especially with ribavirin, has been shown to eliminate the virus from TC plants (Busogoro, Vandermolen, Masquelier, & Jijakli, 2006)

3.5. Banana virus X

BVX is an unassigned member of the family *Betaflexiviridae* and has been detected mostly in symptomless *Musa* plants. The occurrence of the virus

was first reported from Guadeloupe and it is known to be widespread in *Musa* germplasm collections, thus suggesting wide distribution (Teycheney, Marais, Svanella-Dumas, Dulucq, & Candresse, 2005). A nested PCR-assay has been established for virus detection (Teycheney et al., 2007). BVX is spread along with the planting material and no insect vector has been identified.

4. CONCLUSIONS

Banana and plantain are high priority crops in the developing countries because of their contribution to dietary energy, nutrition, and income for the millions of resource-poor farmers who grow over 85% of the world's banana. During the last decade, banana and plantain production around the world increased by 27% (FAOStat, 2012), indicating the high demand. The fruit are particularly valued in resource-poor agriculture because they yield, irrespective of the seasons. Viral, bacterial, and fungal pathogens and nematodes pose a particular concern as they can be moved through planting materials between fields and across borders. Virus disease not only causes yield reductions but is also a major constraint to the exchange of germplasm.

So far, eight viruses have been reported to infect *Musa* spp. Out of these, BBTV and BBrMV and the species complex, BSVs, are significant threats to banana and plantain. Although reports are from a few countries, it is widely believed that BSVs are distributed worldwide, especially in areas where banana and plantain with B genome composition are grown. All these viruses are spread around the world mainly through germplasm. Occurrence of BBTV has expanded markedly during the past 15 years, particularly in SSA, because many countries do not adhere to safe procedures for germplasm exchange.

Recommendations given in the Technical Guidelines for the Safe Movement of *Musa* Germplasm published by the FAO and the International Board for Plant Genetic Resources (IBPGR) in 1996 (Diekmann & Putter, 1996) provide useful information on best practices for the exchange of germplasm and the procedures for disease indexing available at that time. The International Transit Centre (ITC), established in 1985 at the Katholieke Universiteit, Leuven in Belgium, has been serving as intermediary to accept germplasm of unknown health status to generate virus-free planting material for international distribution after checking at one of the two virus indexing centers based at QDPI (Australia) and at CIRAD (France). Despite the existence of such a facility, because of poor awareness,

germplasm continues to be moved across porous borders, especially in SSA, as part of the traditional exchanges or for sale in local markets.

Many countries in Asia, Oceania, and Latin America have devised the large-scale production of virus-free planting material as a measure to contain virus spread and also to prevent yield losses in endemic areas. Although data on the effectiveness of this model in reducing disease impact are limited to Australia, similar measures are slowly gaining popularity because of the widespread epidemics of banana bacterial wilt disease in East Africa.

Efforts are also being made to (i) enhance capacity for the production and supply of virus-free planting material of farmer-preferred cultivars in BBTD-affected countries; (ii) increase vigilance in regions bordering BBTD-affected areas for pre-emptive action to be taken against the introduction of infected planting materials; (iii) demonstrate eradication programs and their benefits; (iv) improve monitoring capacity; and (v) deploy rapid eradication responses in mitigating the spread of the disease. Efforts on the development of host plant resistance through PDR are currently receiving high priority because of its potential to incorporate virus resistance in any cultivar.

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