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A possible scenario for the evolution of *Banana streak virus* in banana

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ABSTRACT

Outbreaks of *Banana streak virus* (BSV) have been recorded worldwide where *Musa* spp. is grown during the last 20 years with no convincing evidence of epidemics. Epidemics were previously reported in Uganda where BSV is currently endemic. BSV is a plant pararetrovirus of the family *Caulimoviridae*, genus *Badnavirus* it causes chlorosis leaf streak disease. The information currently available on banana streak disease makes it possible to identify a complex of distinct BSV species each causing the same disease. BSV exists in two states: one as an episomal form, infecting plant cells; the other as viral DNA integrated within the B genome of banana (endogenous BSV-eBSV) forming a viral genome for *de novo* viral particles. Both forms can be infectious in banana plants. The BSV phylogeny is polyphyletic with BSV distributed in two clades. Clade 1 clusters BSV species that occur worldwide and may have an eBSV counterpart, whereas Clade 3 only comprises BSV species from Uganda. Clearly, two distinct origins explain such BSV diversity. However, the epidemiology/outbreaks of BSV remains unclear and the role of eBSV needs to be clarified. In this review, the biodiversity of BSV is explained and discussed in the light of field and molecular epidemiology data. A scheme is proposed for the co-evolution of BSV and banana based on old or recent infection hypotheses related to African domestication sites and banana dissemination to explain the disease context.

1. Introduction

Plant viruses are intra-cellular parasites that occur widely in intimate association with their hosts. Their diversity in cultivated plants is the result of virus evolution driven by a combination of environmental selective forces and human activity. Virus invasion of cultivated plants likely first occurred around 8000 years ago in the eight global crop domestication centers proposed by Vavilov (1926) (Harlan, 1971, 1981; Lovisolo et al., 2003a,b). The interfaces between natural vegetation and cultivated areas are likely to have facilitated host shifts of indigenous viruses from natural wild ancestors in primitive agricultural systems (Jones, 2009). Most viruses have deleterious effects on cultivated plants as well as on native wild plants and cause diseases with varying economic impacts on crops. As one of the main means for infecting other plants, insect vectors contribute to natural field transmission and to the spread of disease, which can result in epidemics. Vegetative multiplication of crop plants can also play a role in the dissemination of viruses from infected plants in places where this technique is still the main way of plant propagation after domestication. This is so for banana (*Musa* spp.) where the crop is propagated either by use suckers or more recently by *in vitro* plantlets because the selected edible banana plants are usually seedless. Mass propagation by *in vitro* culture is still the most appropriate way to supply large quantities of pathogen- and pest-free *Musa* planting material. However, in this way, all the viruses encountered in banana can also be propagated both horizontally and vertically. Among viruses described from banana, *Banana streak virus* (BSV) of the family *Caulimoviridae*, genus *Badnavirus*, is the most widely distributed on banana and plantains, although only a few epidemics have been reported. The potential economic impact of such epidemics on banana production is still unclear (Daniells et al., 2001; Lassoudière, 1974), as is our ability to find BSV-free banana plants.

BSV is a plant pararetrovirus, which causes chlorosis leaf streak disease. It is a double-stranded circular DNA virus 7.2 to 7.8 kbp long, and uses a virus-encoded reverse transcriptase (RT)

Abbreviations: BSV, *Banana streak virus*; eBSV, endogenous *Banana streak virus*; BSOLV, Banana streak *Obino l'ewai* virus; BSGFV, Banana streak *Goldfinger* virus; BSIMV, Banana streak *Imové* virus; BSMYV, Banana streak *Mysore* virus; BSVNV, Banana streak *Vietnam* virus; BSCAV, Banana streak *Cavendish* virus; BSPEV, Banana streak Peru virus; BSAcYUV, *Banana streak acuminata Yunnan virus*; BSUAV, Banana streak Uganda A virus; BSUIV, Banana streak Uganda I virus; BSUJV, Banana streak Uganda J virus; BSUKV, Banana streak Uganda K virus; BSULV, Banana streak Uganda L virus; BSUMV, Banana streak Uganda M virus; PKW, pisang klutuk wulung; EAH, East African Highland.

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to replicate. It is encapsidated into bacilliform particles and infects other plants by 'horizontal' semi-persistent mealybug-mediated transmission. In contrast to animal retroviruses, badnaviruses do not require integration to replicate. The viral genome contains three open reading frames (ORFs) on one strand. ORFs I and II potentially encode two small proteins of 20.8 and 14.5 kDa, respectively, of unknown function. ORF III encodes a polyprotein of 220 kDa encompassing a putative cell-to-cell movement protein, a coat protein, an aspartic protease, a reverse transcriptase (RT), and a ribonuclease H (RNaseH) (Harper et al., 1999; Hull, 2002). This polyprotein is likely to be cleaved into functional units by the aspartic protease following full-length translation.

There is a high degree of heterogeneity among BSV isolates, which display pronounced variability both serologically and genetically (Geering et al., 2000; Lockhart and Olszewski, 1993). Lockhart and Olszewski (1993) identified at least five distinct serological isolates from Morocco, Rwanda, Trinidad, Honduras and Philippines. Geering et al. (2000) compared the RT and RNase H regions of four Australian isolates with one from Nigeria and demonstrated that they differed by 21.8% to 33.6% in sequence from each other. Such divergence in this conserved region enabled the classification into distinct BSV species according to ICTV criteria (Fauquet et al., 2005; King et al., 2012) for species demarcation in the genus *Badnavirus*. However, all BSV isolates reported worldwide produce the same characteristic intermittent symptoms of discontinuous chlorotic streaks that become necrotic, and a split of the pseudo-stem. In addition, a broad range of variable symptoms, ranging from none to plant death, has been described depending on the virus isolate, host cultivar, and environmental conditions concerned (Dahal et al., 1998, 2000; Lockhart and Jones, 2000).

The transmission of BSV by mealybugs does not appear to be affected by the biological differences observed between vectors species. Experimental transmissions were first undertaken with *Planoccocus citri* and *Pseudoccocus* spp. but no clear relationship between BSV species and mealybug species has been established. Several species of mealybugs are reported to transmit BSV in the field, mainly according to geographic location of the mealybug populations. *Dysmicoccus* spp. is reported in West Africa and South America, whereas *Planoccocus musa* is reported in BSV-infected fields in Nigeria (Dahal et al., 2000; Matile-Ferrero and Williams, 1995). Field observations suggest the virus spread by mealybug transmission is slow (Daniells et al., 2001).

The first banana streak virus outbreaks occurred in the Nieky Valley in Ivory Coast in 1958 (Lockhart and Jones, 2000) in the "Gros Michel" triploid *Musa acuminata* (AAA) banana cultivar. The viral origin was established later (Yot-Dauthy and Bové, 1966) following a severe attacks recorded in Ivory Coast. In 1986, Lockhart reported the disease in southern Morocco in field-grown Dwarf Cavendish bananas (AAA) and confirmed the viral etiology by purifying bacilliform viral particles (Lockhart, 1986). Meanwhile, sporadic outbreaks occurred in Nigeria, Cameroon, Zanzibar, Tanzania and Rwanda that affected different banana genotypes mainly those harboring the B genomes. However infections affected only a few banana and plantain plants in villages and incidence did not exceed 17% (Gauhl et al., 1999; Lockhart and Jones, 2000; Stover, 1988). Similar observations were made in Central and South America, where BSV incidence was reported in plantain (AAB) in the Sula Valley in Honduras and in the Los Rios province of Ecuador (Jones, 2000). In addition the "Mysore" cultivar (AAB) was shown to be chronically infected by BSV. In Australia, BSV was first detected in 1992 in the *Musa* group cv. "Mysore" (Thomas et al., 1994) and since then, the most severe epidemics have been in the Cavendish cultivar (AAA) infected by four BSV species named *Banana streak OL virus* (BSOLV), *Banana streak CA virus* (BSCAV), *Banana streak MY virus* (BSMYV), and *Banana streak GF virus* (BSGFV) (Daniells et al., 2001; Geering et al., 2000).

In the last 20 years, banana streak disease has been reported worldwide where *Musa* spp. are grown but with no convincing evidence of epidemics. Many spontaneous disease outbreaks have been recorded over the years in many banana-producing countries illustrating the worldwide distribution of BSV. In parallel to the common horizontal BSV transmission, many reports described an alternative vertical transmission of this virus due to the presence of homologous integrated BSV sequences within the *Musa balbisiana* genomes (Chabannes et al., 2013; Gayral et al., 2008; Harper et al., 1999; Iskra-Caruana et al., 2010; LaFleur et al., 1996; Lheureux et al., 2003; Ndowora et al., 1999). These endogenous BSV sequences (eBSVs) arose from illegitimate recombination between the virus and the plant genome likely during repair of plant DNA breaks (Gayral et al., 2008; Liu et al., 2012). eBSVs can escape spontaneously following known stress conditions such as use of *in vitro* culture or interspecific breeding processes (Chabannes et al., 2013; Cote et al., 2010; Dallot et al., 2001; Gayral et al., 2008; Lheureux et al., 2003; Ndowora et al., 1999). They reconstitute *de novo* infectious particles possibly through two-step intra-strand homologous recombination (Iskra-Caruana et al., 2010; Chabannes and Iskra-Caruana, 2013), thereby releasing a viral genome causing subsequent exogenous infections. As with BSV, these viruses can contribute as their exogenous counterpart to plant infection and consequently cause disease. The recent spread of BSV has been associated with the diffusion of micro-propagated interspecific *Musa* hybrids (AAB and AAAB) harboring infectious eBSV in their B genomes. Four naturally widespread BSV species have so far been regularly identified as infectious integrants arising from the *M. balbisiana* genome: BSOLV, *Banana streak IM virus* (BSIMV), BSMYV, and BSGFV (Gayral et al., 2008; Geering et al., 2000; Iskra-Caruana et al., 2010).

No epidemics have yet been confirmed worldwide apart from those restricted to East Africa recorded in Uganda in 1996, where BSV is currently described as endemic (Kubiriba et al., 2001b; Tushmereirwe et al., 1996). Currently, all the information available on banana streak disease has made it possible to identify a complex of distinct BSV species that each causes the same disease. BSV exists in two states: an episomal form that infects plant cells, and as eBSV within the B genome of banana. Both forms can be infectious in banana plants. However, the epidemiology of BSV is still unclear and the role of eBSV needs to be clarified. Both field and molecular epidemiology could provide insights into the ecology and evolution of these viruses and help understand these complex interactions. In this review, the biodiversity of BSV is described and discussed in the light of field and molecular epidemiology data in order to account for BSV and banana co-evolution.

2. BSV genetic diversity

Several studies have characterized the genetic diversity of BSV. Harper et al. (2005) described for the first time the diversity of BSV from sequences isolated from field-grown banana of the East African Highland banana group (EAH AAA) (Karamura and Karamura, 1996). They described a polyphyletic BSV phylogeny, structured in three clades (named 1, 2 and 3) based on analyses of a partially conserved region of ORF III encoding the RT and RNase H proteins (Harper et al., 2002, 2004). To obtain an overview of the distribution of BSV, a phylogeny was inferred based on full-length genome sequences from both the 12 available BSV species and the twenty-five badnaviruses representative of the diversity of the genus (Fig. 1). In contrast to the results of Harper et al. (2005), we found all BSV species to be distributed over Clades 1 and 3, and none in Clade 2. Clade 1 clustered BSV species collected worldwide and interestingly, Clade 3 only contained BSV species isolated from Uganda (Gayral and Iskra-Caruana, 2009; Iskra-Caruana et al.,