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Subviral agents associated with plant single-stranded DNA viruses

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Abstract

Begomoviruses (family *Geminiviridae*) are responsible for many economically important crop diseases worldwide. The majority of these diseases are caused by bipartite begomovirus infections, although a rapidly growing number of diseases of the Old World are associated with monopartite begomoviruses. With the exception of several diseases of tomato, most of these are caused by a monopartite begomovirus in association with a recently discovered essential satellite component (DNA-β). These begomovirus/satellite disease complexes are widespread and diverse and collectively infect a wide variety of crops, weeds and ornamental plants. Non-essential subviral components (DNA-1) originating from nanoviruses are frequently associated with these disease complexes, and there are tantalizing hints that further novel satellites may also be associated with some begomovirus diseases. DNA-β components can be maintained in permissive plants by more than one distinct begomovirus, reflecting less stringent requirements for *trans*-replication that will undoubtedly encourage diversification and adaptation as a consequence of component exchange and recombination. In view of their impact on agriculture, there is a pressing need to develop a more comprehensive picture of the diversity and distribution of the disease complexes. A greater understanding of how they elicit the host response may provide useful information for their control as well as an insight into plant developmental processes.

Contents

Plant single-stranded DNA viruses

Only two families of plant-infecting single-stranded (ss) DNA viruses are recognized at this time, the *Geminiviridae* and the *Nanoviridae*. Crop diseases that have since been attributed to geminiviruses, for example, cassava mosaic and maize streak diseases in Africa and beet curly top disease in California, were reported over 100 years ago (Bock, 1982), and geminiviruses continue to have a worldwide impact on agriculture to this day. Indeed, the first description of a geminivirus disease may well have been recorded as early as 752 AD in the Man'yoshu, a classical anthology of Japanese poetry. A poem attributed to the Empress Koken described the autumnal appearance of eupatorium plants in the summer, an observation which has been linked with yellow vein disease of this perennial shrub (Inouye and Osaki, 1980) that has recently been shown to be caused by a geminivirus/satellite disease complex (Saunders et al., 2003). Although the diseases caused by geminiviruses represent serious constraints to agriculture, little was known about the causal

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agents of the diseases until the isolation of virus particles with a unique twinned quasi-isometric morphology associated with maize streak and beet curly top diseases (Bock et al., 1974; Mumford, 1974). This attribute provided the name geminivirus, from Gemini, the sign of the zodiac symbolized by twins (Harrison et al., 1977), and has remained a unifying feature of this family of viruses. Recent structural analysis has demonstrated that the 22 × 38 nm particles associated with *Maize streak virus* (MSV) consist of two incomplete $T = 1$ icosahedra (Zhang et al., 2001), and a similar structure was subsequently observed for *African cassava mosaic virus* (ACMV) (Böttcher et al., 2004). In groundbreaking research, Harrison et al. (1977) and Goodman (1977a) demonstrated that the so-called geminate particles associated with cassava latent virus, MSV and bean golden mosaic virus contained circular ssDNA, and that this genomic DNA was infectious when re-introduced to plants by mechanical inoculation (Goodman, 1977b), setting geminiviruses apart from all other plant viruses that had been characterized at that time. Evidence was provided for *Bean golden mosaic virus* and *Tomato golden mosaic virus* (TGMV) to suggest that at least some geminiviruses had divided genomes (Haber et al., 1981; Bisaro et al., 1982; Hamilton et al., 1982). Shortly afterwards, the nucleotide sequence of cassava latent virus (subsequently renamed ACMV) was established, and infectious clones were used to demonstrate a bipartite genomic structure (Stanley, 1983; Stanley and Gay, 1983). Subsequently, the monopartite geminiviruses MSV, *Beet curly top virus* (BCTV) and *Tomato pseudo-curly top virus* were similarly characterized (Howell, 1984; Mullineaux et al., 1984; Grimsley et al., 1987; Stanley et al., 1986; Briddon et al., 1996), resulting in the present-day recognition of four genera (*Mastrevirus*, *Begomovirus*, *Curtovirus* and *Topocuvirus*) in the family *Geminiviridae* by the International Committee on Taxonomy of Viruses (ICTV) (Stanley et al., 2005). Geminivirus components vary in size between 2500 and 3100 nucleotides, and each encodes two or more genes that are distributed between both the virion-sense and complementary-sense DNA strands and are transcribed bidirectionally from an intergenic region which also contains the origin of replication. The leafhopper-transmitted mastreviruses are quite distinct from all other geminiviruses in terms of their genomic organization and a collective host range that is largely confined to monocotyledonous plants (reviewed by Palmer and Rybicki, 1998). In contrast, the genomic organization of the whitefly-transmitted begomoviruses, leafhopper-transmitted curtoviruses and the treehopper-transmitted topocuvirus is very similar, differing only slightly in gene content and function, indicative of a more recent common evolutionary origin in dicotyledonous hosts. There are now 133 officially recognized geminivirus species of which 117 belong to the genus *Begomovirus* (Stanley et al., 2005), and there are almost 400 complete nucleotide sequences deposited in databases (Fauquet and Stanley, 2005), reflecting their economic importance and enormous diversity resulting from their widespread geographic distribution and host adaptation.

Although many begomoviruses have two genomic components, referred to as DNA-A and DNA-B (Fig. 1), only single components (equivalent to DNA-A) have been reported for a large number viruses. In many cases, this may reflect the relative difficulty in detecting and isolating the more diverse DNA-B component. However, unambiguous evidence was provided to demonstrate that isolates of tomato yellow leaf curl virus (subsequently reclassified as two distinct species *Tomato yellow leaf curl virus* (TYLCV) and *Tomato yellow leaf curl Sardinia virus* (TYLCSV)) and *Tomato leaf curl virus* (ToLCV) had only monopartite genomes (Kheyr-Pour et al., 1991; Navot et al., 1991; Dry et al., 1993). DNA-A encodes the

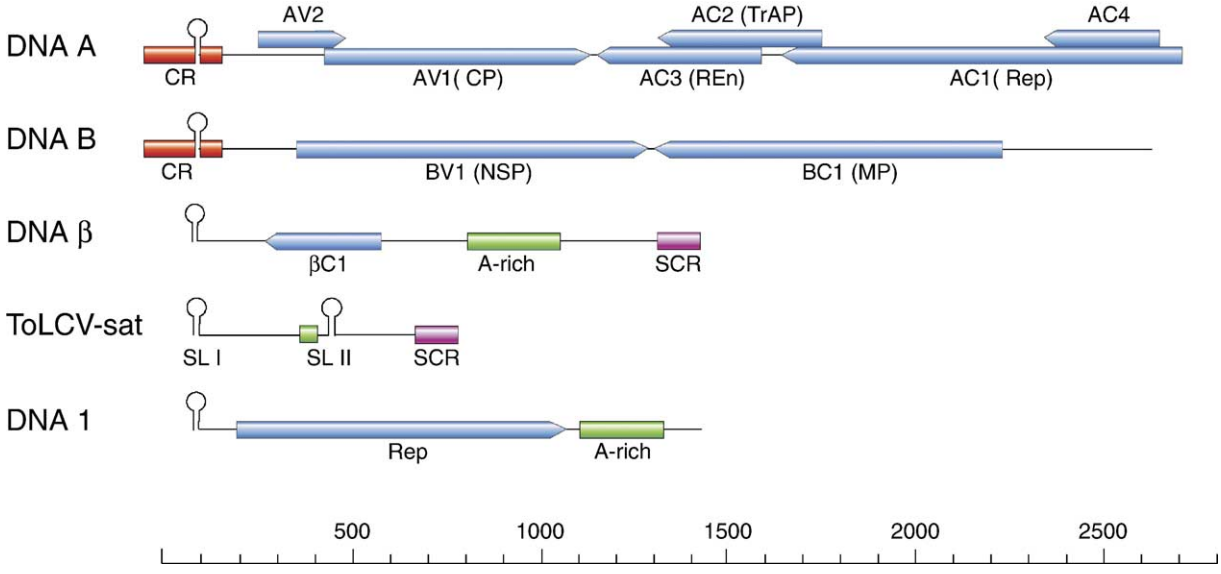

Fig. 1. Genome organization of begomoviruses and their associated subviral components. All components are circular but are shown as linear maps for clarity. The common region (CR) is highly conserved between DNA-A and DNA-B components of a particular begomovirus. The satellite-conserved region (SCR) is highly conserved between all known DNA-β components, and a slightly modified version occurs in ToLCV-sat. Genes encoding the coat protein (CP), replication-associated protein (Rep), transcriptional transactivator protein (TrAP), replication enhancer (REn), movement protein (MP) and nuclear shuttle protein (NSP) are indicated.

coat protein (CP), replication-associated protein (Rep) and its auxiliary replication enhancer protein (REn), and transcription transactivator protein (TrAP) involved in the control of both viral and host gene expression. Some begomoviruses also encode AV2 and AC4 proteins that participate in virus movement and cell-cycle control. The DNA-B component encodes a movement protein (MP) and nuclear shuttle protein (NSP) required for movement of the virus within and between cells (reviewed by Hanley-Bowdoin et al., 1999; see also the review by D. Bisaro in this issue). The absence of a DNA-B component associated with TYLCV, TYLCSV and ToLCV may be attributable to differences in DNA-A gene functions, tissue specificity and adaptation of these viruses to a permissive host in which the DNA-B encoded movement functions are redundant. Despite intensive research into geminivirus diseases by many groups worldwide over a large number of years, the aetiology of the many presumed monopartite begomoviruses remained unexplored until recently.

Although diseases associated with nanoviruses have also been recognized for some time, for example, banana bunchy top disease was first reported in 1889 (Stover, 1972), the precise aetiology has remained elusive owing to the complexity of nanovirus genomes. As a consequence, the molecular characterization of nanoviruses has somewhat lagged behind that of the geminiviruses. Like geminiviruses, nanovirus particles were shown to contain circular ssDNA, although this is encapsidated in small (17–20 nm) isometric particles (Randles et al., 1987; Chu and Helms, 1988; Harding et al., 1991). All viruses within the family *Nanoviridae*, recently adopted by the ICTV, are transmitted by aphids and have been classified into two genera. Those that infect dicotyledonous plants (*Milk vetch dwarf virus*, *Subterranean clover stunt virus* and *Faba bean necrotic yellow virus*) are included in the genus *Nanovirus* while the genus *Babuvirus* contains a single member, *Banana bunchy top virus* (Vetten et al., 2005). Nanoviruses have adopted an entirely different coding strategy to geminiviruses in which each component generally encodes just a single product on the virion-sense DNA strand, although a small number of components may encode two products (Beetham et al., 1997). Nanovirus components are much smaller that those of geminiviruses, ranging in size from 1000 to 1100 nucleotides. At least eight distinct nanovirus components have been isolated that encode Rep (DNA-R), coat protein (DNA-S), cell-cycle link protein (DNA-C), movement protein (DNA-M) and nuclear shuttle protein (DNA-N) as well as proteins of unknown function (Burns et al., 1995; Katul et al., 1998; Sano et al., 1998; Vetten et al., 2005). In addition to the DNA-R component which encodes the so-called master Rep responsible for *trans*-replication of all bona fide components, nanovirus diseases are also associated with a variable number of phylogenetically distinct Rep-encoding components that are distinguishable from DNA-R components by subtle differences in the arrangement of their control sequences. These components are capable of autonomous replication but play no role in *trans*-replication of any other component and rely on the accompanying nanovirus components to provide gene functions necessary for their proliferation (Timchenko et al., 1999, 2000; Horser et al., 2001a, 2001b). They have no known biological function and, in view of their erratic presence, probably play no essential role in maintenance of the disease. As they are clearly related to DNA-R components, they cannot be considered as satellite or satellite-like DNAs according to current definitions (Mayo et al., 2005) but instead should be considered simply as deficient DNA-R components (referred to here as DNA-Rd). The exact number of essential nanovirus components remains unknown and awaits the construction of a full complement of infectious cloned components and the demonstration of transmission of the cloned virus and accompanying disease between plants using the aphid vector in order to confirm Koch's postulates.

Geminiviruses replicate by a rolling circle mechanism (Saunders et al., 1991; Stenger et al., 1991) from a double-stranded (ds) DNA intermediate, produced by complementary-sense DNA synthesis that is initiated from a short RNA primer (Donson et al., 1984; Saunders et al., 1992). In addition, a recombination-dependent replication strategy has been invoked to explain the production of certain replication intermediates associated with the begomoviruses *Abutilon mosaic virus* (AbMV), ACMV, TYLCV, ToLCV and the curtovirus BCTV (Jeske et al., 2001; Preiss and Jeske, 2003; Alberter et al., 2005). In view of their ssDNA genomes and known gene functions, it is generally assumed that nanoviruses replicate in a similar manner. Although de novo DNA synthesis is achieved by recruiting host-encoded factors, initiation of replication is mediated by Rep nicking within a highly conserved loop sequence (TAATATTAC for geminiviruses (Laufs et al., 1995a; Heyraud-Nitschke et al., 1995; Stanley, 1995) and TAT/GTATTAC for nanoviruses (Hafner et al., 1997; Timchenko et al., 1999)) which, together with small reiterated flanking sequences (referred to as iterons) and intervening sequences, form the origin of virion-sense DNA replication. The iterons are highly sequence-specific Rep binding sites that participate in the initiation of replication as well as the control of complementary-sense gene expression (reviewed by Hanley-Bowdoin et al., 1999). Rep binding at the iteron located between its promoter and coding sequence serves to down-regulate its own expression (Eagle et al., 1994). It has been proposed that iteron binding occurs prior to the introduction of a nick within the virion-sense strand of the loop sequence (Laufs et al., 1995a; Orozco and Hanley-Bowdoin, 1996), covalent attachment of Rep to the exposed 5′-terminus (Laufs et al., 1995b) and elongation of the 3′-terminus using the complementary-sense template following the recruitment of host factors (Nagar et al., 1995; Kong et al., 2000; Luque et al., 2002; Selth et al., 2005). The sequence-specific nature of the high-affinity binding site explains why Rep and the origin of replication of distinct begomoviruses are usually incompatible. While this will serve to maintain the genome integrity of bipartite begomoviruses, it is well documented that DNA-A is highly recombinogenic, having the propensity to donate its origin of replication to other components, thus encouraging diversification (Roberts and Stanley, 1994; Hou and Gilbertson, 1996; Stanley et al., 1997; Saunders and Stanley, 1999; Saunders et al., 2002a).

Discovery of subviral agents associated with begomovirus diseases

Satellites are defined as viruses or nucleic acids that depend on a helper virus for their replication but lack extensive nucleotide sequence homology to the helper virus and are dispensable for its proliferation (Mayo et al., 2005). Satellite viruses encode a structural protein that encapsidates its own nucleic acid while satellite nucleic acids rely on the helper virus structural protein for encapsidation and do not necessarily encode additional non-structural proteins. A third type of agent, referred to as satellite-like nucleic acid, also depends on the helper virus for its replication but provides a function that is necessary for the biological success of the helper virus and is therefore considered as being part of the helper virus genome. The first satellite RNA was identified in 1969 in association with the nepovirus *Tobacco ringspot virus* (Schneider, 1969), and since that time, a large number of satellite RNAs, associated with several groups of plant viruses, have been reported (Mayo et al., 2005). The vast majority of these satellite RNAs do not encode functional proteins but nevertheless can have a dramatic effect on the symptoms induced by their helper viruses, ranging from symptom amelioration to an increase in symptom severity (Roossinck et al., 1992).

The first begomovirus satellite to be discovered, referred to as ToLCV-sat, was isolated from tomato plants infected with the monopartite begomovirus ToLCV (Dry et al., 1997). The circular satellite is small (682 nucleotides), has no extensive open reading frames and has little sequence similarity to its helper virus with the exception of sequences within the apex of two stem-loop structures, one containing the ubiquitous geminivirus TAATATTAC motif and the other containing a putative ToLCV Rep binding motif (Behjatnia et al., 1998). ToLCV-sat is not required for ToLCV infectivity and has no effect on the symptoms induced by the helper virus but is dependent on the helper begomovirus for its replication and encapsidation and hence has the hallmarks of a satellite DNA.

Ageratum conyzoides (ageratum) is a widespread weed that frequently exhibits a yellow vein phenotype (ageratum yellow vein disease; AYVD) that has been attributed to geminivirus infection (Tan and Wong, 1993; Wong et al., 1993). The monopartite begomovirus *Ageratum yellow vein virus* (AYVV) was isolated from infected ageratum, and the single genomic component was shown to be infectious in *Nicotiana benthamiana* (Tan et al., 1995). However, re-introduction of the cloned genomic component into ageratum produced only an asymptomatic infection (Saunders and Stanley, 1999; Saunders et al., 2000) suggesting that, in contrast to ToLCV, another factor was required to restore pathogenicity in the natural host. During the search for additional viral components, a number of small circular recombinant components, each containing the AYVV origin of replication together with sequences of unknown origin, were isolated from infected ageratum (Stanley et al., 1997). When co-inoculated into *N. benthamiana*, the recombinants behaved as defective interfering DNAs by ameliorating the symptoms of AYVV infection and reducing the accumulation of the helper begomovirus. Similar recombinants were also identified for the begomoviruses associated with cotton leaf curl disease (CLCuD) (Liu et al., 1998; Briddon et al., 2000, 2001). The significance of the unidentified sequences within the recombinants was not appreciated at the time, but they were to provide a vital clue in the discovery of a new class of satellites.

CLCuD was reported in the Sudan almost 75 years ago and has been a major constraint to cotton production in Pakistan for over a decade (Nour and Nour, 1964; Briddon and Markham, 2000). The disease is associated with several distinct monopartite begomoviruses and recombinants (Zhou et al., 1998; Idris and Brown, 2002; Briddon, 2003; Mansoor et al., 2003; Kirthi et al., 2004). As was observed for AYVV, the cloned genomic component of cotton leaf curl virus (renamed *Cotton leaf curl Multan virus* (CLCuMV)) failed to produce typical CLCuD symptoms, suggesting the presence of another factor (Briddon et al., 2000). The search for additional components resulted in the isolation of a small circular ssDNA, referred to as DNA-1 (Mansoor et al., 1999), that is representative of a new class of component associated with monopartite begomoviruses that have adopted this nomenclature (Briddon et al., 2004). CLCuD DNA-1 is approximately half the size of its helper begomovirus (1370 nucleotides) and encodes Rep (Fig. 1). It can replicate autonomously although it depends on the helper begomovirus for encapsidation and movement both within and between plants. It is clearly related to nanovirus Rep-encoding components and most closely to the above-defined DNA-Rd components (Saunders et al., 2002b; Briddon et al., 2004). It has been suggested that DNA-Rd components became associated with begomoviruses during mixed infections, necessitating a slight size increase in order to be compatible with their new helper virus. It is known that begomoviruses have a stringent size surveillance mechanism that operates during virus movement within the plant (Etessami et al., 1989; Elmer and Rogers, 1990; Gilbertson et al., 2003), and it is possible that size constraints also apply during encapsidation (Frischmuth et al., 2001). Comparison of DNA-1 and DNA-Rd components suggests the increase in size of the nanovirus component may have occurred by inclusion of an A-rich region, possibly generated by a template slipping mechanism during replication. Despite sharing no significant sequence homology with their helper begomoviruses and dependence on the begomovirus for their maintenance, DNA-1 components cannot strictly be defined as satellite DNAs according to current guidelines because they replicate autonomously (Mayo et al., 2005). In view of their clear evolutionary origin, they could be considered simply as either nanovirus-like components or begomovirus-adapted nanovirus components. However, the fact that they play no essential role in proliferation of the helper begomovirus yet appear to be frequently associated with begomovirus diseases (Briddon et al., 2004) suggests that a new category of satellite component is necessary.

Coconut foliar decay virus is an unassigned member of the family *Nanoviridae* that differs from all other nanoviruses in that it is planthopper transmitted, and only a single component has been isolated (Randles et al., 1987; Rohde et al., 1990). At

approximately 1300 nucleotides, the Rep-encoding component is significantly larger than other nanovirus components. Phylogenetic analysis shows that it clusters with DNA-Rd components (Saunders et al., 2002b), suggesting that it might represent a DNA-1 component that has become adapted to a monocotyledonous host by association with a geminivirus, possibly representing an entirely new genus of planthopper-transmitted geminiviruses, or some other pathogenic agent.

Although the discovery of CLCuD DNA-1 did not resolve the aetiology of cotton leaf curl disease, it represented another important step in the search for an additional component. A database search showed that previously undefined sequences within a recombinant associated with AYVV infection (Stanley et al., 1997) originated from a similar nanovirus-like component, leading to the isolation of AYVD DNA-1 from ageratum (Saunders and Stanley, 1999). However, the fact that undefined sequences in the majority of AYVV recombinants were related to each other but entirely unrelated to nanovirus-like components initiated the search for their putative parental component. In this way, a novel ssDNA component of approximately half the size of the helper begomovirus was isolated and shown to induce the yellow vein phenotype when re-introduced with AYVV into ageratum (Saunders et al., 2000). The component was named DNA-β because, in many respects, it functionally resembled the DNA-B component of bipartite begomoviruses. Transmission of both components and propagation of the disease in ageratum using the whitefly vector confirmed the aetiology of the disease. Soon afterwards, a DNA-β homologue isolated from cotton was used to show that CLCuD was caused by a similar monopartite begomovirus/DNA-β complex (Briddon et al., 2001), and since then, many more such complexes have been identified in a wide variety of plant species growing throughout Africa and Asia (Mansoor et al., 2001; Amin et al., 2002; Briddon et al., 2003; Jose and Usha, 2003; Saunders et al., 2003; Shih et al., 2003; Zhou et al., 2003a, 2003b; Bull et al., 2004; Cui et al., 2004a, 2004b; Jiang and Zhou, 2004; 2005; Rouhibakhsh and Malathi, 2005; Were et al., 2005a, 2005b; Wu and Zhou, 2005; Xiong et al., 2005). ToLCV-sat shows significant homology with DNA-β components, for example, it shares approximately 45% nucleotide sequence identity with the component from ageratum. This suggests that ToLCV-sat is a defective satellite that has remained in association with ToLCV despite playing no role in the proliferation of the helper begomovirus, and that ToLCV may once have been associated with a functional DNA-β component prior to its adaptation to tomato (Saunders et al., 2000). This raises the intriguing possibility that the monopartite begomoviruses TYLCV and TYLCSV may also have been associated with such a component before adaptation to this host.

DNA-β components share no significant homology to their helper begomoviruses on which they are dependent for their replication, encapsidation and movement within and between plants, for which reason they have been officially designated as satellite DNAs (Mayo et al., 2005). However, although AYVV and CLCuMV can systemically infect ageratum and cotton, respectively (Saunders and Stanley, 1999; Briddon et al., 2000), and AYVV is whitefly transmissible between ageratum plants (Saunders et al., 2000), both helper begomoviruses accumulate only to low levels in these hosts in the absence of DNA-β. For this reason, it is likely that the helper begomovirus will not be maintained in its natural host in the absence of a DNA-β component. Although this awaits verification, it is arguable that DNA-β is an essential component of the disease complex and as such should be regarded as a satellite-like DNA that is an integral part of the viral genome according to current guidelines. It is recognized that the distinction between satellite and satellite-like nucleic acids can be very slight (Mayo et al., 2005).

While DNA-1 components clearly derive from nanoviruses, the evolutionary origin of DNA-β remains unclear. DNA-β components contain an A-rich region (Fig. 1) suggesting that, like DNA-1, they may have originated as a bona fide component of another pathogenic agent prior to being captured by the begomovirus, necessitating a slight increase in size. However, such a putative pathogenic agent need not necessarily have existed in plants. For example, ToLCV has been show to replicate in agrobacterium, a soil-borne prokaryote that can transfer exogenous DNA into plants where it becomes integrated into the genome (Rigden et al., 1996). This observation implies that similar fundamental processes occur in prokaryotic and eukaryotic backgrounds, prompting the suggestion that geminiviruses may have originated from prokaryotic episomal replicons that undergo rolling circle replication. Intriguingly, AbMV DNA has been shown to be associated with plastids as well as the nucleus (Gröning et al., 1987), an observation that may reflect a past functional relationship with these putative prokaryotic-like endosymbionts. Phylogenetic analysis provides compelling evidence to suggest that vertebrate circoviruses may have originated from plant nanoviruses (Gibbs and Weiller, 1999), possibly facilitated by arthropod vector intermediaries. Hence, it is not inconceivable that genetic material can also be transferred in the opposite direction, to plants from animals or sap-sucking arthropods.

Two novel subviral DNAs (referred to as satDNA-II and satDNA-III) originating in Tanzania have recently been isolated from cassava infected with bipartite begomoviruses (C. M. Fauquet, personal communication). They are relatively small (approximately 1000 and 1200 nucleotides) and contain a GC-rich region, yet are distinct from each other (23% nucleotide identity) and from all geminiviruses and other subviral components. As they have been isolated only from symptomatic plants, they may represent a novel type of satellite DNA that is adapted to bipartite begomoviruses. Although nothing is yet known about their replication and gene expression strategies, infectivity studies have demonstrated that satDNA-II and satDNA-III intensify symptoms in cassava caused by ACMV, *East African cassava mosaic virus* and *East African cassava mosaic Cameroon virus*, and allow these begomoviruses to produce symptomatic infections in an otherwise resistant cassava landrace (TME3). This calls for a more detailed analysis of these intriguing components and an assessment of their contribution to the cassava mosaic disease

pandemic caused by these viruses that is currently affecting many central and east African countries (reviewed by Legg and Fauquet, 2004).

Replication and compatibility of begomovirus subviral agents

Comparison of the growing number of DNA-β components has indicated that they have a highly conserved structure (Fig. 1). In addition to the abovementioned A-rich region, DNA-β components encode a single gene (βC1) and contain a highly conserved sequence of approximately 80 nucleotides, referred to as the satellite-conserved region (SCR). PCR primers based on this sequence have provided a simple and robust method for the isolation of DNA-β components (Briddon et al., 2002). The SCR is located adjacent to the putative stem-loop structure which contains the TAA/GTATTAC motif which, by analogy to geminiviruses, is the site where Rep introduces a nick during the initiation of virion-sense DNA replication. The fact that naturally occurring mutants lacking the βC1 coding region (Briddon et al., 2003) and mutants in which the βC1 coding region and A-rich region have been deleted in vitro (Tao et al., 2004; Qian and Zhou, 2005) are maintained by the helper begomovirus is consistent with the involvement of the SCR and stem-loop in replication, although the identification of precise DNA-β sequences that contribute to replication awaits fine mapping studies. Two-dimensional gel electrophoresis analysis of ToLCV and CLCuD DNA-β replication intermediates suggests that the satellite replicates using similar rolling circle and recombination-dependent replication mechanisms (Alberter et al., 2005). However, analysis of ToLCV-sat suggests that these satellites have adopted a mechanism that is subtly different to that used by bipartite begomoviruses. The observation that ToLCV-sat proliferated in the presence of ACMV and BCTV (Dry et al., 1997) was unexpected because neither virus has the precise ToLCV and ToLCV-sat high-affinity Rep binding motifs that should be necessary for *trans*-replication according to the generally accepted model for the initiation of replication. The idea that this class of satellite component may be rather a promiscuity was reinforced by the isolation of a single CLCuD DNA-β component in association with several distinct begomoviruses in the field (Mansoor et al., 2003). Furthermore, AYVD and CLCuD DNA-β components can be maintained by CLCuMV and AYVV, respectively, and alter the helper virus phenotype in the permissive host *N. benthamiana*. However, neither combination can produce a symptomatic infection in ageratum, implying that host-specific factors are encoded by both the begomovirus and its satellite (K. Saunders and J. Stanley, unpublished data). AYVD DNA-β can also be maintained in plants by *Sri Lankan cassava mosaic virus* (SLCMV), a bipartite begomovirus that is quite distinct from AYVV with respect to sequence and host range (Saunders et al., 2002a). This interaction not only altered the host range of SLCMV to include ageratum but also allowed the helper to dispense with its DNA-B component, implying that the DNA-β component is functionally analogous to the bona fide begomovirus component. Because the DNA-A component can functionally interact with DNA-β and produces a symptomatic infection in *N. benthamiana* in the absence of a DNA-B component, it was suggested that SLCMV was originally a monopartite begomovirus that captured a DNA-B component from *Indian cassava mosaic virus*, in doing so altering its host range to include cassava.

The ToLCV high-affinity Rep binding site is located adjacent to the stem-loop containing the nick site, as has been found for other begomoviruses. Comparison with DNA-β components shows that ToLCV-sat contains a slightly modified SCR at this position, but the binding site is located outside of this region, within stem-loop II (Fig. 1). Interestingly, inverted repeat sequences are also found at the same relative position in other DNA-β components, suggesting that they may be involved in Rep binding. Indeed, deletion of these sequences prevents AYVD DNA-β *trans*-replication by AYVV (K. Saunders and J. Stanley, unpublished data) although, unlike ToLCV-sat, the AYVD DNA-β component does not contain the helper begomovirus putative Rep binding motif (Saunders et al., 2000). In contrast to all other reports for begomoviruses, it has been demonstrated that the binding sites are not essential for ToLCV and ToLCV-sat proliferation in tomato (Lin et al., 2003), indicating that high-affinity binding is not prerequisite for replication, at least for this monopartite begomovirus and its satellite. However, it is possible that a more transient interaction involving the binding site, a cryptic version of the motif or an entirely unrelated sequence, is necessary to correctly position Rep within the origin. The apparently indispensable nature of the high-affinity binding site for replication of bipartite begomoviruses (Fontes et al., 1994a, 1994b) suggests that it may have evolved to ensure that both genomic components are stably maintained. While it remains to be seen if these binding sites are generally dispensable for monopartite begomovirus/satellite complexes, this less stringent requirement may account for the observed replicational promiscuity of satellite components.

DNA-1 components have been found in association with several distinct monopartite begomoviruses that have been isolated from a range of plant species growing in different regions throughout Africa and Asia (Mansoor et al., 1999, 2000a, 2000b, 2001; Saunders and Stanley, 1999; Briddon et al., 2004; Wu and Zhou, 2005). Phylogenetic analyses indicate that they form a cluster that is distinct from components associated with nanoviruses but show no clear grouping according to geographic location or host range, suggesting that they may be relatively mobile rather than adapted to a particular begomovirus species (Saunders et al., 2002b; Briddon et al., 2004). However, DNA-1 components are noticeably absent from begomovirus/DNA-β complexes associated with honeysuckle yellow vein mosaic disease and eupatorium yellow vein disease, both of which occur in Japan. Whether these diseases became geographically isolated prior to the capture of a DNA-1 component or a component was originally present but subsequently lost as a consequence of host adaptation may possibly be resolved by a more thorough investigation of the aetiology of these diseases in Southeast Asia. Although they are frequently found in association with

monopartite begomovirus/DNA-β complexes, there have been no reports to suggest that DNA-1 components occur naturally in association with bipartite begomoviruses. However, infectivity studies have demonstrated that ACMV and TGMV, representatives of Old and New World bipartite begomoviruses, respectively, can maintain AYVD DNA-1 in *N. benthamiana* (Saunders and Stanley, 1999; Saunders et al., 2002b). Adaptation from aphid-transmitted nanoviruses to whitefly-transmitted begomoviruses would undoubtedly have lifted constraints imposed by insect host-preference and brought DNA-1 into contact with potentially new hosts. This was highlighted by the demonstration that AYVD DNA-1 readily became adapted to sugar beet and leafhopper transmission when associated with the curtovirus BCTV (Saunders et al., 2002b), suggesting that DNA-1 components may not necessarily be confined to monopartite begomovirus diseases. However, attempts to adapt the component to the mastrevirus *Bean yellow dwarf virus* in *N. benthamiana* were unsuccessful, implying incompatibility with one or more of the helper virus gene products, which probably reflects divergence of mastreviruses from begomoviruses and curtoviruses.

Functions of begomovirus subviral agents

Begomovirus/DNA-β disease complexes induce a variety of host-specific symptoms ranging from a vein yellowing phenotype in ageratum, eupatorium and honeysuckle, that has no obvious adverse effect on plant survival (Wong et al., 1993; Saunders et al., 2003; Were et al., 2005a), to severe leaf curl, chlorosis and stunting in crops such as cotton, tobacco and mungbean, that can have a significant effect on productivity (Harrison et al., 1997; Briddon and Markham, 2000; Cui et al., 2004b; Rouhibakhsh and Malathi, 2005). Initial studies to resolve the aetiology of AYVD and CLCuD clearly demonstrated that the DNA-β components made an important contribution to the disease phenotype (Saunders et al., 2000; Briddon et al., 2001). Integration of AYVD DNA-β into the *N. benthamiana* genome resulted in severe developmental abnormalities in transgenic plants, indicating that the component encodes a pathogenicity determinant that is functional in the absence of the helper begomovirus (Saunders et al., 2004). Transcript mapping identified a single gene (βC1) on the complementary-sense strand of AYVD DNA-β with the potential to encode a protein of approximately 14 kDa (Saunders et al., 2004), and a similar genetic organization was subsequently shown for CLCuD DNA-β (Saeed et al., 2005). Circumstantial evidence that DNA-β pathogenicity is attributable to βC1 activity was provided by the analysis of naturally occurring CLCuD DNA-β mutants in which the entire βC1 coding region was absent (Briddon et al., 2003). These mutants were *trans*-replicated by the helper virus and moved systemically throughout the plant but were unable to induce typical disease symptoms. The analysis of AYVD and CLCuD DNA-β mutants in which the βC1 coding region had been disrupted (Saunders et al., 2004; Saeed et al., 2005) and expression of βC1 from a transgene in *N. benthamiana* and *N. tabacum* (Cui et al., 2005a; Saeed et al., 2005) provided direct evidence for the involvement of this gene in symptom induction. Interestingly, AYVD DNA-β mutants in which the first in-frame methionine codon of βC1, located nine nucleotides downstream of the major transcript start, had been replaced with a nonsense codon, were still able to produce attenuated symptoms in ageratum (Saunders et al., 2004), suggesting that an alternative initiation mechanism may be used for βC1 expression under these circumstances. Having identified βC1 as the only gene product encoded by DNA-β, it was realized that many of the previously described AYVD recombinants (Stanley et al., 1997) retained the intact coding region. Not only do these recombinants co-exist with DNA-β in the field, but they are able to support a mild symptomatic infection in ageratum in the absence of DNA-β when co-inoculated with AYVV (Saunders et al., 2001). The mild symptoms were associated with reduced AYVV accumulation, suggesting that the begomovirus/recombinant combination would be at a disadvantage compared to the normal begomovirus/DNA-β complex and so less likely to be maintained in the field. Nonetheless, it is interesting to draw a comparison between the recombinants and DNA-B components of bipartite begomoviruses. Having received the intergenic region from the helper virus, the recombinants effectively have acquired a common region (Fig. 1) and, without prior knowledge of the existence of DNA-β components, could have been mistaken for bona fide begomovirus components. It is not inconceivable that a more virulent or better host-adapted recombinant could actually displace the DNA-β component to produce a novel type of disease complex.

Despite having a profound effect on symptom development, little is known about the function(s) of βC1. AYVD DNA-β does not significantly affect AYVV replication in an *N. benthamiana* leaf disk assay although it does enhance the systemic accumulation of the helper begomovirus in its natural host ageratum (Saunders et al., 2000). Similarly, CLCuD DNA-β augments the accumulation of CLCuMV in cotton (Briddon et al., 2001). CLCuD βC1 is able to complement the movement of the bipartite begomovirus *Tomato leaf curl New Delhi virus* DNA-A component in the absence of DNA-B, as well as mutants of V1 and C4 encoded by the monopartite begomovirus ToLCV (M. A. Rezaian, personal communication) that have been implicated in virus movement (Rojas et al., 2001). While this suggests the involvement of βC1 in virus movement, nuclear localization of the protein (Cui et al., 2005a, 2005b) argues against a direct role in facilitating movement across the plasma membrane. The host response to βC1 expression (discussed below) suggests that it may be involved in re-programming the infected cell to provide conditions more suitable for begomovirus replication. Alternatively, as gene silencing suppressors frequently induce pathogenic effects in plants (Voinnet et al., 1999), βC1 may function by countering a host defense mechanism. Consistent with this, βC1 encoded by the DNA-β component associated with *Tomato yellow leaf curl China virus* (TYLCCNV) has been shown to suppress post-transcriptional gene silencing, and suppressor activity as well as symptom induction requires βC1 nuclear localization (Cui et al., 2005b). βC1 was also shown to

bind to both ssDNA and dsDNA in a sequence non-specific manner, although how this relates to its function remains to be determined. As for many geminivirus gene products, it is likely that βC1 will prove to be a multifunctional protein.

It is now well established that geminiviruses modify the cell cycle in order to provide a suitable environment to support their replication. This is brought about by the action of replication-associated proteins that bind to plant homologues of the retinoblastoma tumor suppressor protein, thereby removing the block that otherwise prevents terminally differentiated cells re-entering S-phase (Xie et al., 1996; Ach et al., 1997; Kong et al., 2000; reviewed by Hanley-Bowdoin et al., 2004). This activity may result in endoreduplication rather than cell division (Nagar et al., 1995; Grafi et al., 1996). However, C4 proteins encoded by the monopartite begomovirus ToLCV and the curtovirus BCTV are important symptom determinants that induce abnormal phloem cell division (hyperplasia) and a characteristic vein swelling phenotype in infected *N. benthamiana*, *N. tabacum*, tomato and sugar beet plants (Stanley and Latham, 1992; Latham et al., 1997; Krake et al., 1998). As the phenotype can occur in the absence of other viral genes, it is possible that C4 induces ectopic division in undifferentiated cells, for example, cambium cells that normally remain competent for mitosis. Although structurally unrelated, AYVD βC1 has been shown to induce a remarkably similar phenotype when expressed in *N. benthamiana* from either a transgene or a *Potato virus X* vector (Saunders et al., 2004). Furthermore, βC1 encoded by the DNA-β component associated with TYLCCNV has a profound effect on the development of spongy and palisade parenchyma cells when expressed as a transgene in *N. tabacum* (Cui et al., 2005a). In addition, the CLCuMV/DNA-β complex causes cellular re-differentiation, manifested by vein swelling and greening, and the production of leaf-like growths in cotton (Briddon and Markham, 2000). A similar phenotype occurs in hollyhock affected by *Althea rosea* enation virus (renamed *Hollyhock leaf crumple virus*) that has since been shown to be associated with a DNA-β component (Bigarré et al., 2001; Briddon et al., 2003). Infected hollyhock exhibits abnormal cambium cell activity leading to the proliferation of vascular tissues, and spongy parenchyma cells are replaced with palisade parenchyma cells, resulting in a greater abundance of chloroplasts that is responsible for the vein greening phenotype. It remains to be seen if these striking effects on plant development play an essential role in maintenance of the disease or if they are an unnecessary side effect resulting from recent adaptation of these begomovirus/DNA-β complexes to new hosts. Irrespective of their contribution to the disease complex, these begomovirus and satellite genes will undoubtedly provide useful tools with which to probe plant developmental processes.

Although DNA-1 components often accompany begomovirus/DNA-β complexes, they appear to play no essential role in disease maintenance. It has been suggested that DNA-1 components may serve to reduce the accumulation of the helper virus by competing for limiting cellular resources, thereby modulating the pathogenic effects of infection that may otherwise compromise plant-to-plant transmission by the insect vector. In this respect, they would resemble the defective-interfering (DI) DNAs that frequently accompany geminivirus infections. For example, DI DNAs associated with the bipartite begomovirus ACMV (Stanley et al., 1990; Frischmuth and Stanley, 1991) and the curtovirus BCTV (Frischmuth and Stanley, 1994; Stenger, 1994) modulate helper virus accumulation, and a subgenomic-sized AYVD recombinant has been shown to reduce AYVV accumulation in *N. benthamiana* (Stanley et al., 1997). Although AYVD DNA-1 had little effect on the accumulation of AYVV in *N. benthamiana* (Saunders and Stanley, 1999), the component associated with tobacco curly shoot disease (TbCSD) reduced the accumulation of its helper begomovirus *Tobacco curly shoot virus* in this host, resulting in attenuated symptoms during the early stage of infection. However, both DNA-1 components caused a dramatic reduction in the accumulation of their respective DNA-β components in *N. benthamiana*, and TbCSD DNA-1 altered the symptom phenotype in this host (Saunders et al., 2000; Wu and Zhou, 2005). The observation that another TbCSD DNA-1 isolate (showing 75% sequence identity) did not alter the phenotype in this way raises the possibility that DNA-1 interaction with the begomovirus/DNA-β complex may be isolate-specific. Nonetheless, the effect that DNA-1 has on viral DNA accumulation, particularly on the levels of DNA-β that encodes a pathogenicity determinant, is consistent with a role in disease modulation.

Diversity and relationships of DNA-β components

Since they were first identified just 5 years ago, well over one hundred full-length DNA-β sequences have been deposited in databases, giving an indication of the widespread nature and importance of these satellites. The DNA-β components fall broadly into two categories, those isolated from plant species belonging to the family *Malvaceae* and those associated with other families (Briddon et al., 2003; Bull et al., 2004). A phylogenetic dendrogram based on an alignment of 125 DNA-β sequences is presented in Fig. 2. A dendrogram based upon alignment of the βC1 coding sequence produces trees with the same topology but differing branch lengths. The dendrogram shows the DNA-β components to be extremely diverse, showing less than 50% nucleotide sequence identity between the most distinct groups. They appear to have taken distinct evolutionary paths to produce two major clades (Briddon et al., 2003), those infecting plants of the family *Malvaceae* and those infecting plants in other families, consistent with the biological data. However, there is evidence to suggest that some components from malvaceous plants may also occur in non-malvaceous plants. Thus, components of the CLCuD complex have been identified in the non-malvaceous species chilli (pepper) and radish (Mansoor et al., 2000b; Hussain et al., 2003). In addition, DNA-β components that group with those originating from non-malvaceous plants have recently been isolated from *Sida acuta* (family *Malvaceae*) (Xiong et al., 2005). This indicates that although the begomovirus/DNA-β complexes generally discriminate between malvaceous and non-malvaceous plants, they can host adapt if presented with

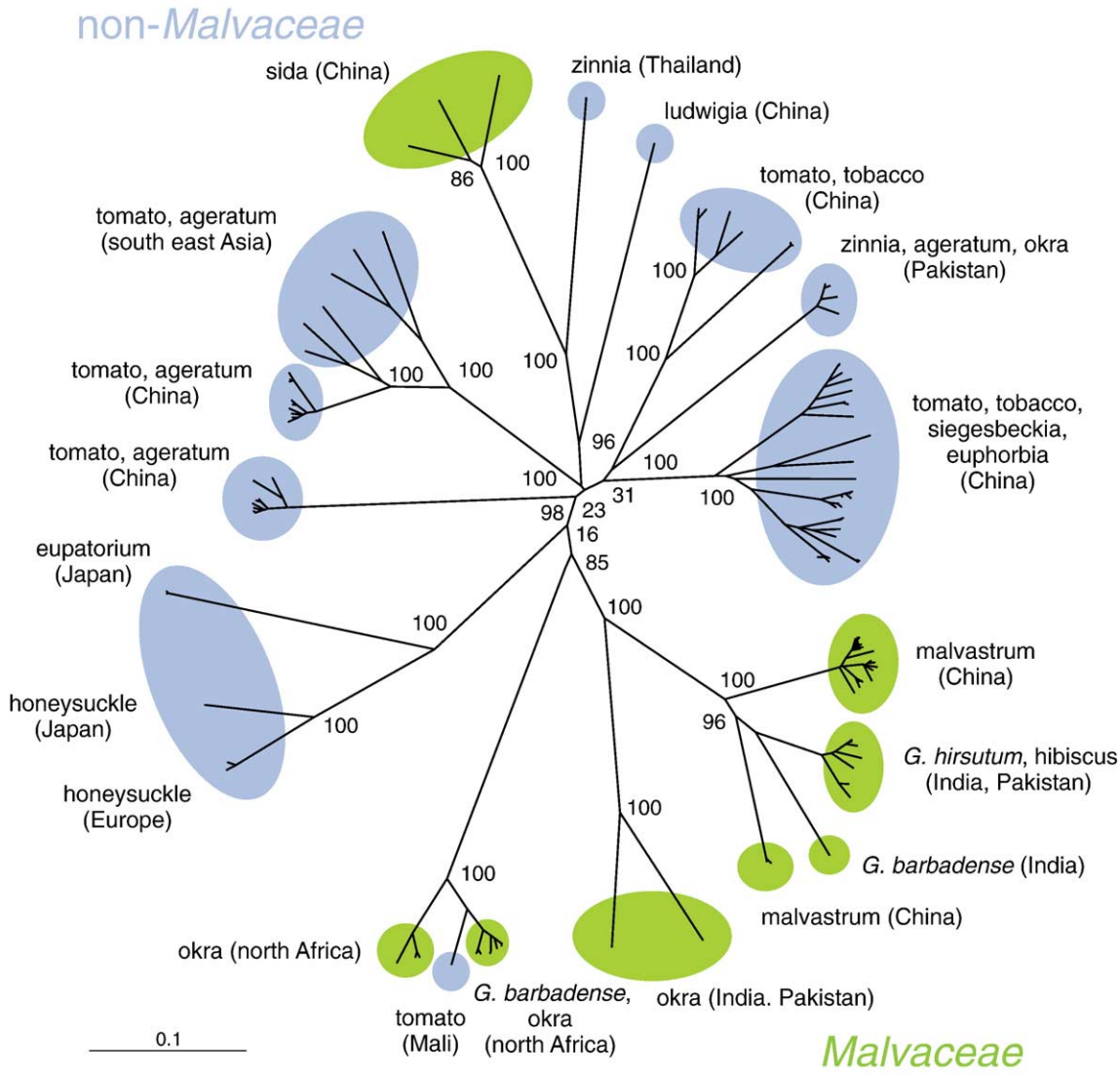

Fig. 2. Phylogenetic dendrogram based upon an alignment of 125 sequences of full-length DNA-β components available in the databases. Numbers at major nodes indicate percentage bootstrap confidence scores (1000 replicates). The geographic origins and the hosts from which the DNA-β components were isolated are indicated.

the opportunity. Alteration of the SLCMV host range to include ageratum by association with AYVD DNA-β (Saunders et al., 2002a) illustrates the important contribution to host adaptation made by the satellite component. The African DNA-β components differ significantly from their Asian counterparts, suggesting diversification as a result of geographic isolation (Idris et al., 2005). With the exception of one DNA-β component isolated from tomato that is closely related to other components from malvaceous hosts, there have been no other examples of DNA-β component being isolated from non-malvaceous plants in Africa. Hence, bearing in mind that only a limited number of samples are available at this time, it is possible that all African DNA-β components originated from malvaceous plants.

Begomovirus/DNA-β complexes associated with CLCuD have received a great deal of attention because of the recent epidemic that has severely compromised cotton production in Pakistan. CLCuD affects *Gossypium hirsutum* and *G. barbadense*, neither of which is indigenous to the Indian subcontinent, having their origins in the New World. As these disease complexes do not occur naturally in the New World, this raises the question as to the origin of CLCuD DNA-β occurring on the Indian subcontinent. Its relatively close relationship to components isolated from *Malvastrum coromandelianum* growing in China (Zhou et al., 2003b; Jiang and Zhou, 2005) suggests that this plant species may have provided the component from which CLCuD DNA-β evolved. Alternatively, the disease may have originated from *Hibiscus rosa-sinensis* which grows throughout the Indian subcontinent and frequently exhibits CLCuD-like symptoms including vein darkening, vein swelling and the formation of leaf-like enations, even in areas not affected by the CLCuD epidemic (Briddon et al., 2003; S. Mansoor and R.W. Briddon, unpublished data). However, as *H. rosa-sinensis* is vegetatively propagated, the begomovirus/DNA-β complex may no longer be readily transmissible from this host which therefore may not act as a

reservoir for the disease. It is interesting to note that a DNA-β component isolated from *G. barbadense* growing in southern India is distinct from that occurring in *G. hirsutum* growing in Pakistan and northern India (77% nucleotide sequence identity), although their βC1 gene products are more closely related (80.5% amino acid identity) which might reflect host adaptation. This suggests that distinct begomovirus/DNA-β complexes may have adapted to cotton at different locations. This idea is supported by the finding that *G. barbadense* in southern India is infected by a distinct begomovirus (Chowda Reddy et al., 2005).

The only begomovirus/DNA-β complex known to occur in the New World is associated with variegated honeysuckle (*Lonicera japonica* variety aureoreticulata). This honeysuckle variety is believed to have been introduced into Europe and North America during the 19th century as an ornamental plant. Fortunately, because plants have been maintained by vegetative propagation in order to retain the yellow vein phenotype, the associated begomovirus/DNA-β complex has lost the ability to be insect transmitted and consequently poses little threat to agriculture. *L. japonica* originates from Asia, and both the begomovirus and DNA-β components have diverged significantly since their introduction to Europe, to the extent that the begomoviruses are now considered to be distinct species (*Honeysuckle yellow vein virus* occurs in the UK and *Honeysuckle yellow vein mosaic virus* in Japan; Stanley et al., 2005). Nonetheless, the DNA-β components from both locations cluster with those isolated from eupatorium growing in Japan (Saunders et al., 2003; Fig. 2), reflecting their original common geographic origin. It remains to be seen if speciation has similarly occurred for components associated with the honeysuckle disease complex in the New World.

Conclusions and future directions

We have only recently become aware that DNA-β components exist, yet they are now recognized as being essential components of disease complexes that pose a threat to agriculture throughout the Old World. The recent isolation of novel satellite components (satDNA-II and satDNA-III) suggests that other disease complexes and subviral components may exist and simply await identification. It should be kept in mind that the majority of geminiviruses have been isolated from symptomatic crop species, and that the many and diverse agriculturally unimportant plant species, both symptomatic and asymptomatic, represent a potentially huge resource which remains largely untapped. It has been shown that both DNA-β and DNA-1 can functionally interact with different begomoviruses and that DNA-1 can even exchange insect vectors by association with a curtovirus. Coupled with the propensity of geminiviruses to undergo recombination and the global movement of both healthy and infected germplasm, this will undoubtedly encourage the dissemination, diversification and host adaptation of these disease complexes. For this reason, a comprehensive assessment of the spatial and temporal distribution and diversity of these complexes in the field is necessary to provide important basic and predictive information for plant health/quarantine organizations as well as breeding programmes. There are regions of the world, including the Americas and Australia, which appear unaffected by these satellite-associated diseases but which cultivate highly susceptible crops and in which the whitefly vector is endemic. Lessons need to be learned from the recent introduction of TYLCV into Central and North America and *Squash leaf curl virus* into the Middle East if we are to avoid unnecessary dissemination of this newly identified class of pathogens. At this time, we do not fully understand the fundamental principles involved in begomovirus Rep-mediated *trans*-replication of DNA-β, hence, a more detailed molecular analysis is necessary to explain why DNA-β components are replicationally promiscuous. There is also a need to investigate βC1 function, to identify viral and host factors with which it interacts and to understand its precise role in symptom development. It is hoped that this will allow a greater appreciation of how and why these disease complexes have such a profound effect on plant developmental processes.

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