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REVIEW ARTICLE

OVERVIEW OF BEGOMOVIRUS GENOMIC ORGANIZATION AND ITS IMPACT

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ABSTRACT

More than 80% of the known geminiviruses are transmitted by whiteflies (*Bemisia tabaci* Gennadius) and belong to the genus *Begomovirus,* which mostly have bipartite genomes designated as DNA-A and DNA-B and infect dicotyledenous plants although numerous begomovirus with a monopartite genome occur in the Old World and there are some of which a single components is not infectious yet no DNA-B component has been found. There have been several of reports of satellite molecules associated with begomoviruses. Genome ORFs are plays important roles for host range determination, virus symptom development & severity, virus movement and virus replication. The frequency with which new begomoviruses are appearing shows that these viruses are still evolving and pose a serious threat to sustainable agriculture, particularly in the tropics and sub-tropics. In recent years, some begomoviruses have also moved to temperate regions causing concern in the production of vegetables in greenhouses. In this review we have discus about the genome organization of begomovirus, its ORFs and their possible pathogenesis n the basis of research findings.

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INTRODUCTION

This is the largest genus in the family *Geminiviridae*. The genus *Begomovirus* is transmitted by whitefly (*Bemisia tabaci* Gennadius) in the persistent circulative manner. *Bean golden mosaic virus* (BGMV) is the type member of the genus. Begomoviruses infect dicots in tropical and temperate climates. The genome of most begomoviruses consists of two ssDNA components, DNA-A and DNA-B, each 2.5-2.8 kb in size. There are two ORFs in the virion sense and four ORFs in complementary sense in DNA-A. DNA-B has one ORF each in virion as well as in complementary sense. In begomoviruses, the tendency for recombination and acquisition of extra DNA components had resulted in emergence of new viruses that infect new hosts and cause new diseases (Varma and Malathi, 2003; Chakraborty *et al*., 2003). The economically most important, geographically wide-spread and numerous geminiviruses are within the genus *Begomovirus* (type species BGMV), which contains more than 200 species (Fauquet *et al*., 2008; Brown *et al*., 1995; Brown and Czosnek, 2002; Jones, 2003; Varma and Malathi, 2003; Brown, 2007; 2010).

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Genome organization of begomoviruses

The International Committee on the Taxonomy of Viruses (ICTV) has recommended a classification and scheme of nomenclature, based on complete nucleotide sequences of the genome (DNA-A and DNA-B) of begomoviruses. Begomovirus originating in the New World have a bipartite genome organization whereas those from the Old World have either bipartite or monopartite genomes (Brown *et al*., 2002). In monopartite begomoviruses such as *Tomato yellow leaf curl virus* (TYLCV) from Israel (Navot *et al*., 1991) and Sardinia (Kheyr-Pour *et al*., 1991), only a single component similar to DNA-A of bipartite begomoviruses in genome organization has been identified (Fig. 1a) and shown to be enough for producing infectivity when reintroduced in tomato, fulfilling Koch's postulates and confirming that the single genomic component is solely responsible for disease development. DNA-A and DNA-B components constitute bipartite genome in begomoviruses. DNA-A is essential for replication and encapsidation (Rogers *et al*., 1986; Townsend *et al*., 1986; Sunter *et al*., 1987) while DNA-B plays a role in systemic movement and symptom production (Etessami *et al*., 1988; Noueiry *et al*., 1994). The begomovirus replication cycles rely entirely on DNA intermediates and occur within the nucleus of the infected cell through two basic stages: conversion of ssDNA to dsDNA intermediates and rolling circle replication (RCR) (Gutierrez, 2002) ((Fig. 1b).

DNA-A of all begomovirus has five ORFs, of which one (AV1, also called AR1) is on the virion DNA strand and the other four (AC1, AC2, AC3 and AC4 also designated as AL1, AL2, AL3 and AL4 respectively) are on the complementary strand. The viral strand ORFs code for the coat protein and for a protein required for cell-to-cell movement of the virus. The proteins encoded by the ORFs on the complementary strand are both involved in viral DNA replication, and are translated from spliced and unspliced versions of the same mRNA. (Harrison *et al.*, 2002) Begomoviruses from the old world posses an additional ORF (AV2) does not found in New World begomoviruses. DNA-A has two ORFs in the virion sense or rightward direction (AV1/AR1-Coat protein (CP) and AV2/AR2-pre coat protein) and five ORFs in the complementary sense or leftward direction (AC1/AL1-replication initiator protein (Rep), AC2/AL2-transcription activator protein (TrAP), AC3/AL3-replication enhancer protein (REn), AC4/AL4 and AC5/AL5). DNA-B has one ORF each in virion strand or right ward direction (BV1/ BR1-nuclear shuttle protein (NSP)) and complementary strand or leftward orientation (BC1/ BL1-movement protein (MP)) (Table 1).

Common region/Intergenic region

DNA-A and DNA-B sequences are different from each other except for an approximately 200 bp intergenic region (IR) (Padidam *et al*., 1995b) and hence that region is also called as common region (CR). The CR is present in the intergenic region between ORFs AV1 and AC1 in DNA-A and between ORFs BV1 and BC1 in DNA-B and it is highly specific for a virus. CR is the only region with significant sequences similarity between DNA-A and DNA-B components of the same virus. The CR has many regulatory element including two TATA motifs, one for ORF AV1/AV2 and another for ORF AC1/AC4. Apparently, the bidirectional promoters for these ORFs are also present in the CR. CR also has a binding site for Rep protein that are repeated (interons), sometimes in perfectly and sometimes in inverted orientation (Arguello–Astoroga *et al*., 1994; Fontes *et al*., 1994). Interon sequences of different viruses vary in length, sequence, number and orientation. There are characteristic differences in the arrangements of the interon between the Old World (Asia, Africa, Mediterranean and Australia) and the New World species (America) (Arguello-Astoroga *et al*., 1994).

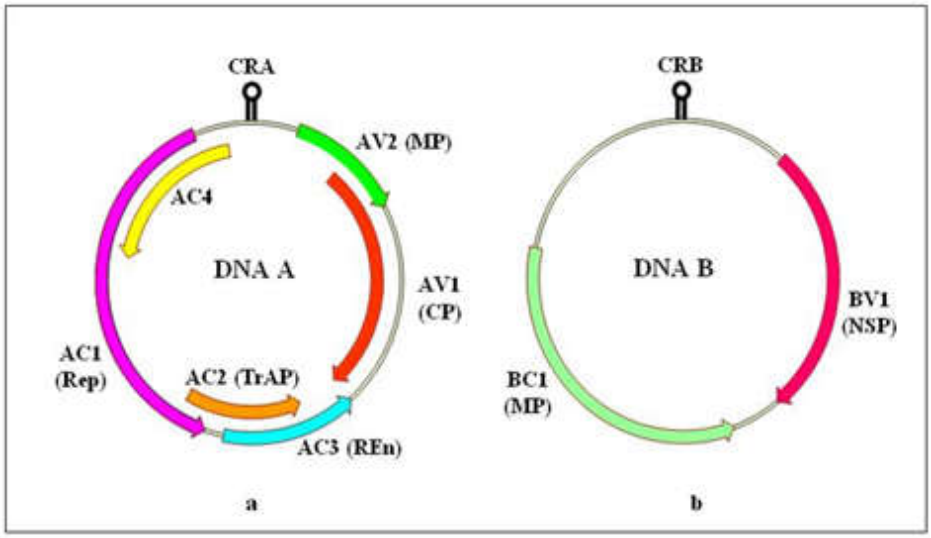

Fig. 1. Genomic organization of begomovirus showing various ORFs (genes) in virion sense and complementary sense: DNA-A (a) and DNA-B (b)

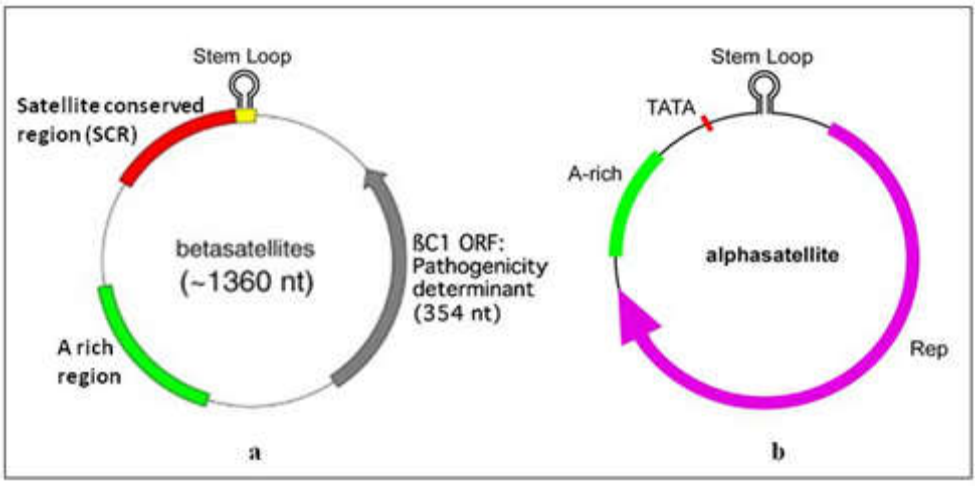

Fig. 2. Genomic structure of betaasatellite DNA component (a) and alphasatellite DNA component Figure source (Xie *et al.*, 2010)

Table 1. ORF (Gene) order in begomovirus DNA-A and DNA-B components with their putative protein products and predicted function

ORF	Predicted molecular weight (kDa)	Putative protein	Predicted function
AV1	30.0	Coat protein (CP)	Encapsidation
AV2	13.0	Pre- coat protein	Cell to cell movement protein
AC1	40.0	Replication initiation protein (Rep)	Replication initiation
AC2	17.0	Transcription activator protein (TrAP)	Transcription activators of rightward ORFs, suppressor of PTGS
AC3	15.0	Replication enancer protein (REn)	Replication enhancement
AC4	6.0	_	Suppressor of PTGS, Viral replication
AC5	18.0	_	Viral replication
BV1	29.0	Nuclear shuttle protein	Nuclear trafficking (NSP)
BC1	32.0	Movement protein (MP)	Cell to cell movement, pathogenicity determinant

Common Region (CR) has the conserved nonanucleotide sequence TAATATTAC present in all geminivirus. The 8th nucleotide, adenine (A) serves as origin of replication (ori). The invariant nonanucleotide sequence present within the loop of stem and loop structure with inverted and complementary stem sequence being variable in composition and length between different viruses (Harrison and Robinson, 1999).

ORF AV1 (Coat Protein)

The ORF AV1 encodes for coat protein (CP) (Kallender *et al*., 1988). In addition to forming the capsid, CP also determines transmission by insect vector. BGMV, the mutation in CP resulted in loss of acquisition and transmission by *Bemisia tabaci* (Azzam *et al*., 1994). In *Abutilon mosaic virus* (AbMV), exchange of three amino acid in the CP of an otherwise non-transmissible isolate resulted in efficient whitefly transmission (Hohnle *et al*., 2001). Trans-encapsidation of *African cassava mosaic virus* (ACMV) genome with *Beet curly top virus* (BCTV), CP allowed ACMV to be transmitted by BCTV leafhopper vector elucidating the role played by CP in insect transmission (Briddon *et al*., 1990). The most conserved region among begomoviruses is the CP gene. This is in conjunction with transmission of begomoviruses by a single species of the vector suggesting the role played by CP in transmission (Stanley, 1985). CP amino acid sequence comparisons first led to conclusion that begomoviruses from the same geographical area are closest in relationship (Hong and Harrison, 1995; Padidam *et al*., 1995b; Rybicki, 1994). The CP gene plays the important role in encapsidation of the viral DNA and is implicated in viral movement within the plant as well as in whitefly transmission (Wartig *et al*., 1997; Hanley-Bowdoin *et al*., 1999; Harrison and Robinson, 1999; Sharma and Ikegami, 2009). It might also play a role in limiting the viral DNA copy number by down-regulating Rep activity, specifically nicking (Yadava *et al*., 2010). In bipartite begomoviruses, the CP, though not needed for mechanical inoculation or agroinoculation of a well adapted host, was needed for whitefly transmission or for agroinoculation in a sub optimal host (Gardiner *et al*., 1988; Pooma and Petty, 1996). For bipartite *Bean dwarf mosaic virus* (BDMV), CP to a limited extent substituted for BV1 function and rescue cell-to cell movement in the absence of ORF BV1 (Sudarshana *et al*., 1998). CP mutants accumulated wild-type levels of dsDNA but only small amounts of ssDNA suggesting that the CP might play a role in sequestering ssDNA (Padidam *et al*., 1996). For monopartite begomoviruses like *Tomato yellow leaf curl virus* (TYLCV), CP was required for infectivity (Ridgen *et al*., 1993). In addition to nuclear import, TYLCV CP was found to play a role in nuclear export acting as a homologue to the bipartite begomovirus BV1 protein (Rojas *et al*., 2001).

ORF AV2 (Pre-coat protein)

Only the Old World begomovirus found to possess ORF AV2. For a bipartite *Tomato leaf curl virus* (TLCV) from India, ORF AV2 had been found to play a role in virus movement (Padidam *et al*., 1996; Wartig *et al*., 1997; Hanley-Bowdoin *et al*., 1999; Harrison and Robinson, 1999; Sharma and Ikegami, 2009) and also as a suppressor of RNA-silencing (Avi *et al*., 2007; Yadava *et al*., 2010). It is speculated that for monopartite begomoviruses found only in the Old World, ORF AV2 is complementing the function of virus movement rendering DNA-B redundant (Harrison and Robinson, 1999).

ORF AC1 (Replication initiator protein)

Replication-associated protein is a multifunctional, oligomeric protein, which possesses site-specific DNA binding to the reiterated motifs (introns) at the intergenic region (IR) and initiates DNA replication by introducing a nick and ligation at the conserved nonanucleotide sequence, executes ATP-dependent topoisomerase I, ATPase and helicase activities and also binding of retinoblastoma-related proteins (Hanley-Bowdoin *et al*., 1999; Harrison and Robinson, 1999; Pant *et al*., 2001; Choudhury *et al*., 2006). Replication initiator protein (Rep) is the only viral protein absolutely essential for replication (Elmer *et al*., 1988). Replication enhancer protein (REn) enhances viral DNA accumulation. This depends on the interaction between REn with Rep, which would bind simultaneously to the stem-loop structure and to the upstream Rep-DNA complex (Hanley-Bowdoin *et al*., 1996). There is no direct evidence for this model but it would serve to direct Rep-DNA complex to the nicking site.

Mutations in REn produced delayed-onset and attenuation of symptoms (Sunter *et al*., 1990). AS mentioned earlier, Rep protein is the only viral protein absolutely required for viral DNA replication as it is responsible for initiating DNA replication during the rolling-circle amplification stage. In all geminiviruses tested so far, Rep has sequence specific DNA binding capacity as well as site specific endonucleolytic activity. Begomovirus Rep interacts with the viral REn (AL3) protein. In all Rep proteins, the C-terminal regions contain a NTP- binding domain with typical walker A and B motifs and exhibits ATPase activity (Gutierrez, 2002). Rep protein (~43 kDa) is multi-functional. During replication, Rep specifically recognizes the viral origin (Fontes *et al*., 1994), binds to specific sequences (repeats/ introns) found in the CR and cleaves the phosphodiester bond between the seventh and eighth residues of the conserved nonamer 5' TAATATTAC 3' (Stanley, 1995).

Rep remains bound covalently to the 5'- phosphate end and the 3'- hydroxyl end becomes available for rolling-circle replication. After one replication cycle, Rep cleaves once again at the newly generated origin sequence. Then Rep ligates the nascent 3' end of DNA with the previously generated 5' end releasing a unit–genome length, circular ss DNA molecule (Bisaro, 1996). Thus, Rep acts as an endonuclease and ligase to initiate and terminate rolling-circle replication (Laufs *et al*., 1995a; Orozco and Hanley-Bowdoin, 1998). The Rep proteins of all geminivirus have an NTP-binding domain (Hanley-Bowdoin *et al*., 1999) and the Rep proteins of *Tomato golden mosaic virus* (TGMV) and *Tomato yellow leaf curl virus* (TYLCV) are known to hydrolyze ATP (Orozco *et al*., 1997; Desbiez *et al*., 1995). Gorbalenya and Koonin (1993) proposed that Rep might function as a DNA helicase during replication. Pant *et al*. (2001) had shown ATP-dependent topoisomerase activity of Rep protein from blackgram isolate of *Mungbean yellow mosaic virus* (MYMV). Rep acts as a transcriptional regulator to regulate its own synthesis (Sunter *et al*., 1993; Eagle *et al*., 1994). Rep has also been shown to oligomerise with itself as well as with REn protein (Settlage *et al*., 1996).

DNA binding, cleaving and ligation domain of Rep is at N' terminus, oligomerization domain is located almost in the middle and ATP hydrolyzing activity at C' terminus (Orozco *et al*., 1997). Sequence analysis of rolling circle initiator proteins of eubacteria, eukaryotes and archaebacteria revealed three-conserved motifs-I (FLTYP), II (HXH) and III (YXXK) (Ilyina and Koonin, 1992). Motif III with a tyrosine (Y) residue is involved in the nucleophilic attack of phosphodiester bond of DNA at *ori* and represents the active site of Rep for DNA cleavage (Laufs *et al*., 1995a). Mutation of the active tyrosine at motif III blocked DNA cleavage and replication by TYLCV Rep protein. Mutation of motifs I, II and III of TGMV Rep protein also blocked DNA cleavage and replication (Orozco and Hanley-Bowdoin, 1998). Two α-helices in the N' terminus of Rep protein, which overlap DNA binding and cleavage domains, were conserved among different geminivirus (Orozco *et al*., 1997). Mutations in these helices inhibited DNA binding and cleavage *in vitro* and viral replication *in vivo*, thereby proving that these helices are essential for Rep function (Orozco and Hanley-Bowdoin, 1998). For TYLCV Rep, the structure of the N' terminus catalytic domain (4 -121 amino acids) had been elucidated by nuclear magnetic resonance (NMR) spectroscopic studies. It comprised nine β-strands and two α-helices (Campos-Olivas *et al*., 2002).

ORF AC 3 (Replication enhancer protein)

Tomato golden mosaic virus (TGMV) Replication enhancer protein (REn) had been shown to interact both with TGMV Rep and with retinoblastoma-related protein (pRBR). The REn oligomerisation was not required for its interaction with Rep or pRBR. Rep and pRBR were found to bind to the same domain in TGMV REn (Settlage *et al*., 2001), although functional relevance of this interaction is not yet clear. It has been proposed that Rep/REn and not pRBR/REn interaction is important for viral DNA replication (Gutierrez, 2002). Recently, Rep proteins from TGMV and *Cabbage leaf curl virus* (CaLCuV) have been shown to interact with *Arabidopsis* proteins-a Ser/Thr kinase-GRIK (Geminivirus Rep-interacting kinase), a kinesin–GRIMP (Geminivirus Rep-interacting motor protein) and a histone H3 protein (Kong and Hanley-Bowdoin, 2002).

ORF AC2 (Transcription activator protein)

Transcription activator protein **(**TrAP) induces virion sense promoters in DNA-A as well as in DNA-B. In TrAP mutants of *Tomato golden mosaic virus* (TGMV), there was no accumulation of coat protein and no infectivity was achieved. Also ssDNA accumulation was impaired just like CP mutation (Sunter *et al.*, 1990). They demonstrated that TrAP transactivates virion sense expression of CP in DNA-A as well as ORF BV1 in DNA-B at transcription level (Sunter and Bisaro, 1991; 1992). Sunter *et al*. (1994) showed that TrAP transactivation was non-specific. TGMV virion sense promoters were transactivated by TrAP protein from other begomoviruses like *African cassava mosaic virus* (ACMV) and *Squash leaf curl virus* (SLCV) but transactivation by TrAP homolog in curtovirus C2 protein was not achieved. TrAP was shown to activate CP by two distinct mechanisms i.e. by activation in mesophyll cells, and derepression in phloem cells (Sunter and Bisaro, 1997). The activation domain was mapped to the C' terminus of the protein (Hartitz *et al.*, 1999). The protein was found in both the nuclear and cytoplasmic compartments when expressed in insect and plant cells (van Wezel *et al.*, 2001). TrAP has anti-gene-silencing determinants as TYLCV-China TrAP protein caused enhanced susceptibility when expressed in transgenic plants (van Wezel *et al.*, 2002a). TrAP was also shown to interact with plant adenosine kinase and SNFI (Sucrose Nonfermenting I) kinase, which are presumed to be components of innate host anti-viral mechanisms (Hao *et al*., 2003), thus explaining enhanced susceptibility shown by TrAP protein transgenics.

The TrAP protein transactivates expression of virion-sense genes from both DNA-A and DNA-B (Hanley-Bowdoin *et al*., 1999; Harrison and Robinson, 1999; Wang *et al*., 2003; Trinks *et al*., 2005; Gopel *et al*., 2007; Pandey *et al*., 2009), inactivates adenosine kinase (ADK) (Wang *et al*., 2003), binds siRNA (Vanitharani *et al*., 2004) and interacts with tomato karyopherin α (Gopel *et al*., 2007). It has also been reported as a suppressor of RNA silencing in bipartite begomoviruses (Voinnet *et al*., 1999; Wang *et al*., 2003; Vanitharani *et al*., 2004; Trinks *et al*., 2005) as well as monopartite begomoviruses (Dong *et al*., 2003; Gopel *et al*., 2007; Kon *et al*., 2007).

ORFs AC4 and AC5

ORF AC4 is present within Rep protein but in a different frame thus it encodes for a different protein. The role of ORF AC4 in the virus infection could not be detected for TGMV (Pooma and Petty, 1996) but later ORF AC4 was presumed to weakly suppress Rep gene expression (Groning *et al.*, 1994) and the binding site for AC4 protein mediated repression did not involve Rep binding site and was distinct (Eagle and Hanley-Bowdoin, 1997). In *Tomato leaf curl virus*, ORF C4 was neither required for the virus replication nor for systemic spread of the virus but is considered to be a determinant of symptom severity (Ridgen *et al.*, 1994). In the case of monopartite *Begomovirus*, TYLCV, it was hypothesized that ORF C4 might play a role in delivering viral DNA to the cell periphery and the plasmodesmata and serve as BCI protein homologue (Jupin *et al.*, 1994; Rojas *et al.*, 2001). Expression of ACMV Rep protein alone induced a local hypersensitive response; co-expression of Rep and AC4 protein triggered systemic necrosis in infected tissues but AC4 protein when expressed alone did not induce any such necrosis (van Wezel

et al., 2002b). It was proposed that ACMV AC4 might counter the plant defense mechanism that was initiated by the Rep-mediated local hypersensitive response. Asymptomatic vein swelling was attributed to AC4 protein activity causing abnormal cell division in vascular bundles (Latham *et al.*, 1997; Krake *et al*., 1998).

The function of AC4 protein is symptom determinant implicated in the control of cell-to-cell movement, and may counter a host response to Rep expression and suppression of RNA-silencing by binding of siRNAs (Wartig *et al*., 1997; Rojas *et al*., 2001; Vanitharani *et al*., 2004; Fondong *et al*., 2007; Gopal *et al*., 2007; Pandey *et al*., 2009). ORF AC5 codes for a protein for which the functional significance is yet to be ascertained. Presence of this ORF is not very frequent. It has also been observed in some geminivirus isolates like *Tomato leaf curl New Delhi virus* (luffa, potato), *Papaya leaf curl virus* (Y15934), *Soybean crinkle leaf virus*- Japan (AB050781), *Croton yellow vein mosaic virus* (AJ507777), *Tomato yellow leaf curl*-Thailand, *Ageratum yellow vein virus* (NC004626), *Euppatorium yellow vein virus*-(Yamaguchi) (AB079766), *Bhendi yellow vein mosaic virus*- (Madurai) (AF63750), *Sida yellow mosaic virus* (NC004639), *African cassava mosaic virus*- (Ivory Costa) (AF259894), and *Water melon chlorotic stunt virus* (AJ245652).

ORFs BV1 (Nuclear shuttle protein) and BC1 (Movement protein)

In general, for bipartite begomoviruses, ORF BV1 functions as a nuclear shuttle protein (NSP) i.e. in transporting the virus DNA between nucleus and cytoplasm, while ORF BC1 (Movement Protein, MP) functions in cell-to-cell movement of the viral DNA (Gafni and Epel, 2002; Hussain *et al*., 2005).
For *Bean dwarf mosaic virus* (BDMV), *Abutilon mosaic virus* (AbMV) and *Squash leaf curl virus* (SLCV), NSP and MP have been shown to function co-operatively in cell-to-cell movement (Rojas *et al.*, 1998; Sanderfoot and Lazarowitz, 1995; Zhang *et al.*, 2001). For BDMV, it was demonstrated that the NSP exports viral DNA from the nucleus to cytoplasm. MP increases the size exclusion limit of plasmodesmata and mediated the cell-to-cell movement of the viral DNA (Rojas *et al.*, 1998). For SLCV, it was suggested that NSP was involved in shuttling viral ss DNA both into and out of the nucleus (Pascal *et al.*, 1994). The NSP-ss DNA complex is trapped by MP and the MP-NSP-ss DNA complex is directed to the cell periphery (Sanderfoot *et al*., 1996). Studies with ACMV did not support the suggestion that NSP and MP function in concert in cell-to-cell spread. However, it was suggested that NSP supported local spread while MP, possibly along with NSP, aided in systemic spread (von Arnim *et al.*, 1993). MP has been shown to be the determinant of pathogenicity of bipartite begomoviruses (von Arnim and Staley, 1992; Pascal *et al*., 1993). The viral disease-like phenotype seen in transgenic plants expressing MP might be due to interference of MP with normal macromolecular intercellular trafficking (Lucas, 1995; 2006) and 3' terminus of movment protein (MP) was associated with symptom development (Pascal *et al*., 1993).

REPLICATION OF GEMINIVIRUS

The geminivirus replication cycle relies entirely on DNA intermediates and occurs within the nucleus of the infected cell through two basic stages:

- Conversion of ssDNA to dsDNA intermediates.
- Rolling circle replication (RCR).

Rolling circle replication

Geminivirus DNA replicates via rolling circle mechanism (RCR) that is analogous to bacteriophage T_4 replication, which also has single-stranded circular DNA. Geminiviruses utilize the host cell machinery for their replication. For initiation of replication, ss DNA of the geminivirus first converted into dsDNA (Kammann *et al.*, 1991; Saunders *et al.*, 1992). This step is carried out entirely by host cellular enzymes. For minus strand synthesis, priming by RNA or DNA is required. For mastreviruses, a small ssDNA molecule, 88 nucleotide-long with ribonucleotides at 5' end was found to be annealed with small intergenic region (SIR) in the encapsidated genomic ssDNA (Donson *et al*., 1984; Hayes *et al*., 1988). For begomoviruses, no such ssDNA primers were found but RNA primers were found in ACMV, associated near the common region (CR) (Saunders *et al*., 1992). The next stage, the rolling circle phase, requires the concerted action of viral Rep protein and other viral proteins with cellular factors and leads to the production of new dsDNA and ssDNA viral forms (Stenger *et al.*, 1991; Stanley, 1995). The double-stranded replicative form serves as a template for transcription as well as ssDNA synthesis. The single-stranded DNA thus produced may enter in the replicative cycle or gets transported from cell-to-cell with the help of viral movement protein or gets encapsidated by viral coat protein (Gutierrez, 2002). The diagrammatic representation of geminivirus replication cycle proposed by Gutierrez (1999). A recombination dependent replication (RDR) mode seen in the late phase of bacteriophage T_4 infection has also been suggested to occur in many geminiviruses like *Abutilon mosaic virus* (AbMV) (Jeske *et al*., 2001) and ACMV, BCTV, TGMV and TYCLV (Preiss and Jeske, 2003).

Transcriptional regulation

Replication (Rep) is known to auto-regulate its own expression. The Rep binding iteron is located between TATA box and Rep transcription start site and when Rep binds to the introns, it down-regulates its own expression (Sunter *et al*., 1993; Eagle *et al*., 1994; Groning *et al*., 1994). Like replication, transcriptional regulation was also specific for the homologous Rep protein. The C2 terminus domain of mastrevirus Rep has a trans-activation domain (Horvath *et al*., 1998), which might be responsible for its transcriptional regulation. For begomoviruses too, the same domain was expected to be responsible for Rep auto-regulation (Gutierrez, 2002). Auto-regulation does not require a functional *viral ori* suggesting that Rep binding site acts independently during transcription and replication (Eagle *et al*., 1994). In TGMV, AL4 mediated repression of AL1 promoter has been reported which does not involve AL1 binding site. Also, a TATA box element located immediately upstream of the Rep binding site and a G-box element located at the base of stem-loop structure is involved in activation of AL1 promoter (Eagle and Hanley-Bowdoin, 1997). Mutations in TATA-box and G-box motifs were detrimental to Rep promoter function. In particular, G-box mutants displayed very low activity indicating that this element is the primary Rep transcriptional activating sequence. TATA-box and G-box element share a role in replication as well as transcription. A host G-box transcription factor might activate Rep expression and facilitate Rep recruitment and

binding to the origin, modulate chromatin assembly and origin accessibility or stabilize an origin conformation required for efficient replication (Hanley-Bowdoin *et al*., 1999). In ACMV, a domain responsible for negative regulation of Rep promoter by its product had been mapped to a 92 bp fragment located immediately upstream of Rep initiation codon encompassing the consensus TATA box and transcription start site (Hong and Stanley, 1995). Like TGMV, ACMV Rep protein could not fully silence its promoter. Though the equivalent sequences are involved in transcriptional regulation of Rep for both ACMV and TGMV, there are some differences too (Hong and Stanley, 1995). ACMV Rep promoter does not have a G-box factor and transcriptional regulation is regulated by multiple *cis*-elements. Also, AC4 protein in ACMV did not negatively regulate Rep expression.

For TGMV and ACMV, a TATA-box containing promoter for TrAP (ORF AC2) expression had been identified (Haley *et al*., 1992; Zhan *et al*., 1991). Neither promoter for TrAP was responsive to Rep or REn protein, ruling out regulation of TrAP promoters by these viral proteins (Haley *et al*., 1992). The virion sense promoters for DNA-A (CP) as well as DNA-B (NSP) in TGMV and ACMV have been mapped. They comprise the CR and downstream sequences that contain TATA box and transcription start site and were enough for virion sense gene activation when TrAP protein was supplied *in* trans (Sunter *et al*., 1990; Sunter and Bisaro, 1991; 1992; Groning *et al*., 1994). Activation of virion sense promoters have been found to occur at the level of transcription (Sunter and Bisaro, 1992). AC2 regulation of virion sense promoter is non-specific, as it has been shown that TGMV DNA-A could complement non-infections ACMV and *Potato yellow mosaic virus* (PYMV) TrAP mutants in plants (Saunders and Stanley, 1995; Sung and Coutts, 1995).

This suggests that TrAP functions either through conserved DNA sequences or through conserved interaction with host factors or both. Within virion sense promoters, a conserved sequence called the conserved late element (CLE), although not ubiquitous, is present in most of the begomoviruses and has been implicated for TrAP interaction (Arguello-Astorga *et al*., 1994). But TrAP interaction with CLE had not been proved yet. Also, when TGMV promoter having CLE and BGMV promoter without CLE were checked for functional equivalence by transactivation with heterologous promoters, TrAP was found to regulate virion sense promoters non-specifically. Thus, the CLE was ruled out in TrAP interaction (Hung and Petty, 2001). TrAP was found to induce CP expression in TGMV by activating it in mesophyll cells and derepressing it in phloem cells (Sunter and Bisaro, 1997). Thus, TrAP induces CP expression by different mechanisms in different cell types by interacting with different components of the cellular transcription machinery. In comparative analysis of complementary and virion sense promoters for ACMV, the complementary sense promoters in DNA-A were found to have stronger activity in comparison with viral sense promoters (Zhan *et al*., 1991). Though upstream sequences of MP (ORF BC1) are similar to that of Rep in the CR, their activation mechanisms are different. In TGMV, 5' transcription start site for MP and Rep are different (Sunter and Bisaro, 1989). Also, Rep did not regulate MP transcription (Sunter *et al*., 1993), indicating that MP has different promoter sequences from that of Rep. Thus, in contrast to virion sense expression, where both genomic components have similar regulation, complementary sense expression is distinct for DNA-A and DNA-B. Thus, in geminivirus transcription, Rep is active in early phase of viral life cycle. When sufficient Rep and AC4 protein accumulates, these promoters are repressed may be in order to limit their interference with the function of downstream TrAP promoters. TrAP and MP promoters are activated midway in viral life cycle. TrAP protein regulate virion sense gene expression and thus CP and NSP are late genes produced late in the viral infectivity cycle. This expression strategy was proposed by Howarth *et al*. in 1985.

Geminivirus infection cycles

The first stage in the infection cycle involves the injection of viral ssDNA into a plant cell by an insect vector. *Geminiviruses* replicate through a double-stranded (ds)DNA intermediate in the nucleus of the infected cells. Upon initial entry of a geminivirus into a host cell, there are no viral proteins present other than CP. Movement to the nucleus must thus be dependent entirely on the CP and exploitation of the host transport mechanism. It is not clear whether the virus inoculated into the host by the vector moves to the nucleus as an encapsulated virion or whether it decapitates and moves as a nucleoprotein complex. Apparently, CP is involved during this transport stage, probably through interactions with the host transport network (Gafni and Epel, 2002). Once in the nucleus, the viral ssDNA is converted into a transcriptionally active dsDNA intermediate that acts as a template for both transcription and replication.

This complementary DNA synthesis is accomplished entirely by host proteins. This viral dsDNA is associated with histones and packaged into so called minichromosomes. Similar to other viral systems, the expression of geminiviral genes seems to follow a finely tuned temporal sequence. It is believed that the genes encoding proteins involved in replication and transcription (e.g. Rep, TrAP, and REn) are expressed earlier than the virion sense genes (e.g. CP and NSP genes). After the expression of the early viral genes (left side or complementary sense), the multiplication of the virus genome by a rolling-circle (RC) mechanism generates new viral ssDNA molecules from the dsDNA intermediate. The last stage of the cycle corresponds to the uptake of the virions by the insect vector. In this case, it has been shown that the CP and, probably, virus particles are indispensable for insect transmission. The viral ssDNA genome replicates in the nucleus via dsDNA templates by a rolling circle mechanism. NSP, the viral-encoded NSP, binds progeny ssDNA genomes and transports these between the nucleus and cytoplasm. MPB, the viral cell-to-cell movement protein, traps NSP-genome complexes in the cytoplasm and directs these to and across the cell wall through modified plasmodesmata. In adjacent uninfected cells, NSP-genome complexes are released, and NSP targets the viral ssDNA to the nucleus to initiate new rounds of replication and infection.

PHYLOGENY OF BEGOMOVIRUSES

For begomoviruses, DNA-A and their conserved protein products CP and Rep are being used for phylogenetic analysis. DNA-B sequences are more diverse than that of DNA-A and there are fewer conserved elements in DNA-B and hence, in general, are not used for phylogenetic analysis (Harrison and Robinson, 1999). When CP and Rep proteins of different begomoviruses were compared, based on geographical origins, they were divided into the Old World (Asian, African,

Mediterranean and Australia) and the New World (North and South America) viruses (Howarth and Vandemark, 1989). This kind of geography-related lineage was confirmed by further analysis including more begomoviruses (Hong and Harrison, 1995; Rybicki, 1994; Padidam *et al*., 1995b). Within the Old World Branch, three sub clusters were discerned for CP: first viruses from African and Mediterranean region, second Asian viruses and third viruses from China and Australia (Harrison and Robinson, 1999). The Old World and the New World division of begomoviruses was further established when complete nucleotide sequence of DNA-A of 120 geminivirus species were analyzed by Fauquet and Stanley (2003). Currently, seven different virus clusters have been discerned for begomoviruses-New World begomoviruses comprising meso and Latin American viruses and viruses infecting sweet potato. The Old World viruses comprising of African, Indian, Asian viruses and viruses infecting legumes (Fauquet and Stanley, 2003). The phylogeny of geminiviruses is extremely stable, even when the number of viruses used for comparison was increased from 36 in 1989 to 389 in 2005. This aspect of stability in the phylogeny of geminiviruses would help in elucidating their evolution.

ASSOCIATION OF SETELLITE MOLECULES WITH BEGOMOVIRUSES

DNA-β molecule

Satellites are the common features of many plant RNA viruses. There are a number of satellite molecules which totally depend upon the associated helper viruses for their replication, transmission, and encapsidation within the same capsid along with the nucleic acid of the helper virus. The satellites have no nucleotide sequence similarity with the associated virus and host genomes (Murant and Mayo, 1982). The both linear and circular types of satellites are found associated with RNA viruses. The majority of satellites interferes with helper virus replication and attenuate disease expression, but there are also a number of satellites known that exacerbate viral symptoms or produce novel symptoms quite distinct from the those induced by the helper virus alone (Collmer and Howell, 1992). Till 1997, there was no any report of association of satellite molecule with plant DNA virus system. Mansoor *et al*. (1999) reported a novel single-stranded circular DNA molecule approximately half the size (1350 nt) of the *begomovirus* component/full length-DNA-A of *Cotton leaf curl virus* (CLCuV DNA-1) associated with cotton leaf curl disease in Pakistan (Fig. 2a).

The nucleotide sequence of this molecule was found to share homology to plant nanoviruses. Later on Mansoor *et al*. (2000b) further reported the association of such type of molecule with the Ageratum yellow vein disease in Pakistan and described that yellow vein disease of ageratum in Pakistan is associated with a begomovirus infection and single-stranded circular DNA molecule with similarity to CLCuV-DNA-1 and termed DNA-β. Since then, a large number of similar disease complexes have been reported from a variety of economically important and weed plant species (Saunders *et al*., 2000; Briddon *et al*., 2001 and 2003; Zhou *et al*., 2003; Mansoor *et al*., 2003; Jose and Usha, 2003; Bull *et al*., 2004; Xiong *et al*., 2005; Rouhibakhsh and Malathi, 2005; Reddy *et al*., 2005; Tahir and Haider, 2005, Snehi et al., 2011, Srivastava et al., 2013a, Srivastava et al., 2013b, Khan and Khan, 2016). Previously the DNA-β were only known to be associated with monopartite begomoviruses (Briddon *et al*., 2003; Jose and Usha, 2003) but now have also been reported with bipartite begomoviruses (Rouhibakhsh and Malathi, 2005; Reddy *et al*., 2005). Beside this there are a numerous report on a nanovirus-like component, termed DNA-1 found associated with the disease complex. DNA-1 components are related to nanovirus components that encode replication-associated proteins. So far they have not been shown to have essential role in the disease etiology. Moreover, the disease plants also frequently contain DNA-β and DNA-1 recombinants that contain the begomovirus origin of replication. The DNA-β satellite components require the helper begomovirus for replication in cells of host plants, systemic infection and encapsidation in virus particles. There is a highly conserved region in their nucleotide sequence adjacent to a ubiquitous stem-loop required for replication, and an extensive adenine-rich region approximately 370 to 420 nts suggested to maintain the size of the component (Briddon *et al*., 2003). All DNA-β molecules encode a conserved open reading frame (ORF) and termed as βCI. The DNA-β shows negligible sequence identity either to DNA-A or DNA-B components associated with begomoviruses except from the sequence TAATATTAC which is common in all geminiviruses which also contain the initiation site of rolling circle replication.

The *βC1* in one instance has been shown to be responsible for the suppression of jasmonic acid signaling involved in at least one gene silencing pathway (Yang *et al*., 2008). Briddon *et al*. (2003) characterized 26 DNA-β molecules from diverse plant species taken/originating from a variety of geographically distinct sources. These molecules were found widely associated with monopartite begomoviruses from Old World and apparently absent from the New World. The phylogenetic analysis of nucleotide sequence data formed two groups; one originated from *Malvaceous* hosts and the second from plants within the *Solanaceae* and *Compositae.* The requirement for a begomovirus and its associated component to produce typical disease symptoms has previously been demonstrated for Bhendi yellow vein mosaic disease (BYVMD, Jose and Usha, 2003), ageratum yellow vein disease (AYVD, Saunders *et al*., 2000) and cotton leaf curl disease (CLCuD, Briddon *et al*., 2001). Saunders *et al*. (2000) found that *Ageratum yellow vein virus* (AYVV) and DNA-β together form a disease complex that is responsible for the yellow vein phenotype of *Ageratum conyzoides*.

The systemic infection of *A. conyzoides* with AYVV alone is asymptomatic and viral DNA replication is reduced to 5% or less of that in presence of DNA-β. Cotton leaf curl disease (CLCuD) in Pakistan caused by *Cotton leaf curl Multan virus* (CLCuMV) was associated with DNA-β component (Briddon *et al*., 2001). The clone of CLCuMV was alone found infectious but not typical to CLCuD. However, when CLCuMV and its respective DNA-β was inoculated together, induced full range of symptoms in experimentally inoculated cotton similar to the naturally infected cotton plants (Briddon *et al*., 2001). Jose and Usha (2003) studied the role of DNA-β molecule associated with the bhendi yellow mosaic disease caused by *Bhendi yellow vein mosaic virus* (BYVMV). The agro-inoculated bhendi plants with only DNA-A did not produce the typical yellow vein mosaic symptoms of the disease. The inoculated plants only showed edge curling of the leaves. However, the clone was found systemically infectious as judged by Southern hybridization. When the bhendi seedlings were inoculated with DNA-A and DNA-β showed

typical yellow vein mosaic symptom. Furthermore, the progeny of the cloned BYVMV DNA-A and DNA-β was found to be whitefly transmitted & the inoculated bhendi plants developed symptoms similar to the disease. These findings strongly support the involvement of the DNA-β components with the disease complex.

Alphasatellites (DNA-1)

The begomovirus/betasatellite complexes are often associated with a second type of circular ssDNA satellite, initially referred to as DNA-1 (Mansoor *et al*., 1999; 2001; Saunders and Stanley, 1999; Briddon *et al*., 2004), but now called alphasatellites (Mubin *et al*., 2009b, Tiendrebeogo *et al*., 2010). Alphasatellites encode a single protein that shares high nucleotide (nt) identity with the replication associated (activator) protein (Rep), a rolling-circle replication initiator protein encoded by viruses in the genus *Nanovirus*, family 26 *Nanoviridae* that also have a genome of circular ssDNA (Fig. 2b) (Gronenborn, 2004; Xie *et al*., 2010). Consequently, alphasatellites are capable of autonomous replication, but require a helper begomovirus for spread in plants and for whitefly vector transmission. In addition to Rep, alphasatellites also have an A-rich region, ~200 nts long, downstream of the Rep-encoding region. Recently, it has been demonstrated that the Rep of the alphasatellite associated with *Tobacco curly shoot virus* (TbCSV) can be used as a virus-induced gene silencing vector (VIGS) (Huang *et al*., 2009). In contrast to betasatellites, alphasatellites possess in their stem loop the nonanucleotide sequence, TAGTATTTAC also found in the stem loop of viruses in the family *Nanoviridae*. Alphasatellites can affect both begomovirus titer and symptom development in host plants (Saunders and Stanley, 1999; Patil and Fauquet, 2010). Initially it was thought satellite molecules were limited to the Old World, but recently, alphasatellites have been found associated with New World begomoviruses (Paprotka *et al*., 2010; Romay *et al*., 2010), thus expanding the geographical distribution of satellite molecules associated with begomoviruses. Some DNA-2 alphasatellites encode a pathogenicity determinant that may modulate begomovirus-betasatellite infection by reducing betasatellite DNA accumulation (Idris *et al*., 2011).

Conclusion

All biological organisms have a genome. As we have seen previously, the virus genome can be either DNA or RNA. This nucleic acid used to encode functions necessary for it to complete its life cycle and its interaction with its environments. There is great variation in the nature of these genomes. Several begomovirus infections have been reported in food legume, vegetable, fibre, ornamental and weed plant species. Begomovirus genome consists of either one or two circular single-stranded DNA components, referred as DNA-A and DNA-B of each about 2.6-2.8 kb in size (Fauquet *et al*., 2008). Each ORFs of these genomes (DNA-A & DNA-B) are plays an important roles for host range determination, virus symptom development & severity, virus movement and virus replication. Monopartite begomoviruses are associated with one or more smaller components, about 1.4 kb in size, known as satellite DNAs molecules. Two types of satellite molecules are known: the alphasatellites and betasatellites, depending upon the organization of their DNA and their effects on the symptoms produced by the helper begomovirus. The alphasatellites it is previously known as DNA-1, encode their own replication-associated protein and are believed to have originated from another class of single-stranded DNA viruses, the nanoviruses (Borah and Dasgupta, 2012). In recent years, some begomoviruses have also moved to temperate regions causing concern in the production of vegetables in greenhouses. Another concern is the emergences of disease that are caused by a complex of begomovirus are satellite DNA molecules. According to the International Committee on the Taxonomy of Viruses (ICTV) recommendation, two sequences belong to two viruses if their complete DNA sequence genome (DNA-A and DNA-B) of begomoviruses is less than 89% identical, and are considered to be variants of the same virus, if the identity is more than 89%. So begomovirus genomic organizations are necessary for proper identification and characterization of begomoviruses at species level on the basis of its genome based phylogenic relationships.

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REFERENCES

Arguello-Astorga, G. R., Guevara-Gonzalez, P.G., Harrera-Estrella, L.R. and Rivera-Bustamante, R.F. 1994. Geminivirus replication origins have a group specific organization of iterative elements: A model for replication. *Virology* 203: 90-100.

Avi, Z., Efrat, G., Levy, Y., Arazi, T., Citovsky, V. and Gafni, Y. 2007. Suppressor of RNA silencing encoded by Tomato yellow leaf curl virus-Israel. *Virology* 358: 159-165.

Azzam, O., Frazer, J., De La Rose, D., Beaver, J.S., Ahlquist, P. and Maxwell, D.P. 1994. Whitefly transmission and efficient ssDNA accumulation of bean golden mosaic geminivirus require functional coat protein. *Virology* 73: 289-296.

Bisaro, D., M. 1996. Geminivirus DNA replication. *In DNA replication in Eukaryotic cell,* pp, 833-854.

Borah B. K., Dasgupta I. 2012. Begomovirus research in India: A critical appraisal and the way ahead. J. Biosci. 37(4), 791–806,

Briddon R.W., Mansoor S., Bedford I.D., Pinner M.S., Saunders K., Stanley J., Zafar Y., Malik K.A. and Markahn P.G. 2001. Identification of DNA β components required for induction of cotton leaf curl disease. *Virology* 285: 234-243.

Briddon, R. W., Bull, S. E., Amin, I., Idris, A. M., Mansoor, S., Bedford, I. D., Dhawan, P., Rishi, N., Siwatch, S. S., Mansoor S., A., M., Brown, J. K., Zafar, Y. and Markham, P. G. 2003. Diversity of DNA β: a satellite molecule associated with some monopartite begomovirus. *Virology* 312: 106-121.

Briddon, R.W., Bull, S.E., Amin, I., Mansoor, S., Bedford, I.D., Rishi, N., Siwatch, S.S., Zafar, M.Y., Abdel-Salam, A.M. and Markham, P.G., 2004. Diversity of DNA 1: a satellite-like molecule associated with monopartite begomovirus-DNA β complexes. *Virology* 324: 462-474.

Briddon, R.W., Pinner, M.S., Stanley, J. and Markham, P.G. 1990. Geminivirus coat protein gene replacement alters insect specificity. *Virology* 177: 628-633.

Brown, J.K. 2010. Phylogenetic biology of the *Bemisia tabaci* sibling species group. Chapter 2 In: P. A. Stansly and S. E. Naranjo (eds.), Bionomics and Management of a Global Pest. *Springer Science,* The Netherlands, pp. 31-67.

Brown, J.K. and Czosnek, H. 2002. Whitefly transmitted viruses. In: *Advances in Botanical Research*, Academic Press, N.Y. pp. 65-100.

Brown, J.K. Idris, A.M. Alteri, C. and Stenger, D.C. 2002. Emergence of a new cucurbit infecting begomovirus species capable of forming viral reassortant with related viruses in the *Squash leaf curl virus* cluster. *Phytopathology* 92: 734-742.

Brown, J.K., 2007. The *Bemisia tabaci* complex: genetic and phenotypic variability drives begomovirus spread and virus diversification. *Plant Disease Feature*, January 2007. http://www.apsnet.org/online/feature/btabaci/.

Brown, J.K., Frohlich, D.R. and Rossell, R.C. 1995. The sweetpotato of silver leaf whiteflies: biotypes of *Bemisia tabaci* or a species complex? *Annals of Entomology* 40: 511-534.

Bull, S.E., Tsai, W.S., Briddon, R.W., Markham, P.G., Stanley, J. and Green, S.K. 2004. Diversity of begomovirus DNA-β satellites of non-malvaceous plants in east and south east Asia. *Arch. Virol*. 149: 1193-1200.

Campos-Olivas, R., Louis J.M., Clerot, D., Gronenborn, B., Gronenborn, A.M. 2002. The structure of a replication initiator unites diverse aspects of nucleic acid metabolism. *Proc. Natl. acad. Sci.* USA 99: 103-105.

Chakraborty, S., Pandel, P.K., Banerjee, M.K. Kallo, G. and Faukuet, C.M. 2003. Tomato leaf curl Gujarat virus, a new begomovirus species causing a severe leaf curl disease of tomato in Vanarisi, India. *Phytopathology* 93: 1485-1494.

Choudhury, N. R., Malik, P. S., Singh, D. K., Islam, M. N., Kaliappan, K. and Mukherjee, S. K. 2006. The oligomeric Rep protein of *mungbean yellow mosaic India virus* (MYMIV) is a likely replicative helicase. *Nucleic Acids Res*. 34: 6362-6377.

Collmer, C.W. and Howell, S.H. 1992. Role of satellite RNA in the expression of symptoms caused by plant viruses. *Annual Review of Phytopathology* 30: 419-442.

Desbiez, C., David, C., Mettouchi, A., Laufs, J. and Gronenborn, B. 1995. Replication protein of tomato yellow leaf curl geminivirus has an ATPase activity required for viral DNA replication. *Proc. Natl. Acad. Sci.* USA, 92: 5640-5644.

Dong, X., Van Wezel, R., Stanley, J. and Hong, Y. 2003. Functional characterization of the nuclear localization signal for a suppressor of posttranscriptional gene silencing. *J. Virol*. 77: 7026-7033.

Donson, J., Morris-Krsinich, B.A.M., Mullineaux, P.M., Boulton, M.I. and Davies, J.W. 1984. A putative primer for second-strand DNA synthesis of maize streak virus is virion associated. *The EMBO Journal* 3: 3069-3073.

Eagle, P., A. and Hanley-Bowdoin, L. 1997. *Cis* elements that contribute to geminivirus transcriptional regulation and efficient DNA replication. *J. Virol*. 71: 6947-6955.

Eagle, P.A., Orozco, B.M. and Hanley-Bowdoin, L. 1994. A DNA sequence required for geminivirus replication also mediate transcriptional activation. *Plant Cell* 6: 1157-1170.

Elmer, J.S., Brand, L., Sunter, G., Gardiner, W.E., Bisaro, D.M. and Rogers, S.G. 1998. Genetic analysis of the *Tomato golden mosaic virus* II. The product of AL1 coding sequence is required for replication, *Nucl. Acids Res.* 16: 7043-7060.

Etessami, P., Callis, R., Ellwood, S., and Stanely, J. 1988. Delimitation of essential genes of cassava latents virus DNA 2. *Nucl. Acids. Res.* 16: 4811-4829.

Fauquet C.M. and Stanley J. 2003. Geminivirus classification and nomenclature Progress and problems. *Annals of Applied Biology* 142: 165-189.

Fauquet, C.M., Briddon, R.W., Brown, J.K., Moriones, E., Stanley, J., Zerbini, M. and Zhou, X. 2008. Geminivirus strain demarcation and nomenclature. *Arch of Virol* 153:783-821.

Fondong, V.N., Chowda Reddy, R.V., Lu, C., Hankoua, B. and Felton, C. 2007. The consensus *N*-myristoylation motif of geminivirus AC4 protein is required for membrane binding and pathogenicity. *Mol. Plant-Microb. Interact*. 20: 380-391.

Fontes, E.P.B., Eagle, P.A., Sipe, P.S., Lucknow, V.A. and Hanley-Bowdoin. 1994. Interaction between a geminivirus replication protein and origin DNA is essential for viral replication. *Journal of Biological Chemistry* 269: 8459-8465.

Gafni, Y. and Epel, B. L. 2002. The role of host and viral protein in the intra and intercellular trafficking of geminivirus. *Physiological and Molecular Plant Pathology* 60: 261-241.

Gardiner, W.E., Sunter, G., Brand, L., Elmer, L.S., Rogers, S.G., and Bisaro, D.M. 1988. Genetic analysis of tomato golden mosaic virus: the coat protein is not required for systemic spread or system development. *EMBO J.* 7: 899-904.

Gopel, P., Kumar, P.P., Sinilal, B., Jose, J., Yadunandam, A. and Usha, R. 2007. Differential roles of C4 and *β*C1 in mediating suppression of post-transcriptional gene silencing: evidence for transactivation by C2 of *Bhendi yellow vein mosaic virus*, a monopartite begomovirus. *Virus Res*. 123: 9-18.

Gorbalenya, A.E. and Koonin, E.V. 1993. Helicase: amino acid sequence comparisons and structure-function relationships. *Curr. Opin. Struc. Biol*. 3: 419-429.

Gronenborn, G. 2004. Nanoviruses: genome organization and protein function. *Vet.Microbiol*. 98: 103-110.

Groning, B.R., Hayes, R.J. and Buck, K.W. 1994. Simultaneous regulation of tomato golden mosaic virus coat protein and AL1 gene expression: expression of the AL4 gene may contribute to suppression of the AL1 gene. *J. Gen. Virol*. 75: 721-726.

Groning, B.R., Hayes, R.J. and Buck, K.W. 1994. Simultaneous regulation of tomato golden mosaic virus coat protein and AL1 gene expression: expression of the AL4 gene may contribute to suppression of the AL1 gene. *J. Gen. Virol*. 75: 721-726.

Gutierrez, C. 1999. Geminivirus DNA replication. *Cell. Mol. Life Sci*. 56: 313-329.

Gutierrez, C. 2002. Strategies for geminivirus DNA replication and cell cycle interference. *Physiological and Molecular Plant Pathology* 60: 219-230.

Haley, A., Zhan, S., Richardson, K., Head, K. and Moris, B. 1992. Regulation of the activities of African cassava mosaic virus promoters by the AC1, AC2, and AC3 gene products. *Virology* 188: 905-909.

Hanley-Bowdoin, L. Settlage, S.B., Orozco, B.M., Nagar, S., Robertson, D. 1999. Geminiviruses: models for plant DNA replication, transcription and cell cycle regulation. *Critical Reviews in Plant Science* 18: 71-106.

Hanley-Bowdoin, L., Eagle, P.A., Orozco, B.M., Robertson, D. and Stellages, S.B. 1996. Geminivirus replication in "*Biology of plant Microbes interaction*" Stacey, G., Mullin, B. Greehof, Eds. St. Paul, M.N., and USA: ASPP, pp. 287-292.

Hao L Wang, H., Sunter, G., and Bisaro, D.M. 2003. Geminivirus AL2 and L2 proteins interact with and inactivate SNFI Kinase. *Plant Cell* 15: 1034-1048.

Harrison, B.D. and Robinson, D.J. 1999. Natural genomic and antigenic variation in whitefly transmitted geminivirus. *Annual Review of Phytopathology* 37: 369-398.

Harrison, B.D., Swanson, M.M. and Fargette, D. 2002. Begomovirus coat protein: serology, variation and function. *Physiological and Molecular Plant pathology* 60: 257-271.

Hartitz, M.D., Sunter, D., and Bisaro, D.M. 1999. The tomato golden mosaic virus transactivator (TrAP) is a single-stranded DNA and zincbinding phosphoprotein with an acidic activation domain. *Virology* 263: 1-14.

Hayes, R.J., MacDonald, H., Coutts, R. H., and Buck, K. W. 1988. Priming of complementary DNA synthesis in vitro by small DNA molecules tightly bound to virion DNA of wheat dwarf virus. *J. Gen. Virol*. 69: 1345-1350.

Hohnle, M. Hofer, P. Bedford, I.D. Briddon, R.W. Markham, P.G. and Frishmuth, T. 2001. Exchange of three amino acids in the coat protein results in efficient whitefly transmission of a non-transmissible *Abutilon mosaic virus* isolate. *Virology* 290: 164-171.

Hong, Y.G. and Harrison, B.D. 1995. Nucleotide sequence from tomato leaf curl viruses from different countries: 'evidence from three geographically separate branches in evolution of the coat protein gene of whitefly transmitted geminiviruses. *J. Gen. Virol*. 76: 2043-2049.

Horvath, G.V., Petlko-szandthner, A., Nikovics, K., Bilgin, M., Boulton, M., Davis, J.W., Gutierrez, C. and Dudits, D. 1998. Prediction of functional regions of the Maize streak virus replication-associated proteins by protein-protein interaction analysis. *Plant Molecular Biology* 38: 699-712.

Howarth, A.J. and Vandemark, G.J. 1989. Phylogeny of geminiviruses *J. Gen. Virol*. 70: 2717-2727.

Howarth, A.J., Caton, J., Bossert, M. and Goodman, R.M. 1985. Nucleotide sequence of bean golden mosaic virus and a model for gene regulation in geminivirus. *Proc. Natl. Acad. Sci.* USA. 82: 3572-3576.

Huang, C., Xie, Y. and Zhou, X. 2009. Efficient virus-induced gene silencing in plants using a modified geminivirus DNA1 component. *Plant Biotechnol. J*. 7: 254-265.

Hung, H.C. and Petty, I.T.D. 2001. Functional equivalence of late gene promoters in bean golden mosaic virus with those in tomato golden mosaic virus. *Journal of General Virology* 82 (3): 667 - 672.

Hussain, M., Mansoor, S. Iram, S. Fatima, A.N. and Zafar, Y. 2005. The nuclear shuttle protein of *Tomato leaf curl New Delhi virus* is a pathogenecity determinant. *Journal of Virology* 79 (7): 4434-4439.

Idris, A. M., Shafiq S. M., Briddon R. W., Khan A. J., Zhu J. K. and Brown J. K. 2011. An unusual alphasatellite associated with monopartite begomoviruses attenuates symptoms and reduces betasatellite accumulation. *Journal of General Virology*, 92, 706–717.

Ilyina, T.V. and Koonin, E.V. 1992. Conserved sequence motifs in the initiator proteins for rolling circle DNA replication encoded by diverse replicons from Eubacteria, Eucaryotes and *Archaebacteria*. *Nucl. Acids. Res*. 20: 3279-3285.

Jeske, H., Lutyeneier, M. and Preib, W. 2001. DNA forms indicate rolling-circle and recombination dependent replication of Abultion mosaic virus. *The EMBO Journal* 20: 6158-6167.

Jones, D.R. 2003. Plant viruses transmitted by whiteflies. *European Journal of Plant Pathology* 109: 195-219.

Jose, J. and Usha, R. 2003. Bhendi yellow vein mosaic disease in India is caused by association of a DNA β satellite with a begomovirus. *Virology* 305: 310-317.

Jupin, I., Kouchkovsky, F. D., Jouanneau, F. and Gronenborn, B. 1994. Movement of tomato yellow leaf curl geminivirus (TYLCV): Involvement of protein encoded by ORF C4. *Virology* 204: 82-90.

Kallender, H., Petty, I.T.D., Stein, V.E., Panico, M. and Blench, I.P. 1988. Identification of the coat protein gene of tomato golden mosaic virus. *Journal of General Virology* 69: 1351-1357.

Kammann, M., Schalk, H.J., Matzeit, V., Accotto, G.P., Crespi, S. and Gorneborn, B. 1991. DNA replication of wheat dwarf virus, a geminivirus, requires two cis-acting signals. *Virology* 184: 786-790.

Khan Z. A., Khan J. A. 2016. Characterization of a new begomovirus and betasatellite associated with chilli leaf curl disease in India. Arch Virol. DOI 10.1007/s00705-016-3096-0.

Kheyr-Pour, A., Bendahmane, M., Matzeit, V., Accotto, G.P., Crespi, S., Gronenborn, B. 1991. Tomato yellow leaf curl virus Sardinia is a whitefly – transmitted monopartite geminiviruses *Nucleic Acids Research* 19: 6763-6779.

Kon, T., Sharma, P. and Ikegami, M. 2007. Suppressor of RNA silencing encoded by the monopartite tomato leaf curl Java begomovirus. *Archives of Virology* 152: 1273-1282.

Kong, Ling-Jie and Hanley-Bowdoin 2002. A geminivirus replication protein interacts with a protein kinase and a motor protein that display different expression pattern during plant development and infection. *Plant cell* 14: 1817-1832.

Krake, L.R., Rezaian, M.A., and Dry, I.B. 1998. Expression of the tomato leaf curl geminivirus *C4* gene produces virus like symptoms in transgenic plants. *Mol. Plant Microbe Interact*. 11: 413-417.

Latham JR, Saudrs K, Pinner MS and Stanley J 1997. Induction of plant cell division by beet curly top virus C4. *The Plant j.* 11: 1273-1283.

Laufs, J., Traut, W., Hayraud, F., Matzeit, V., Rogers, S.G., Schell, J. Gronenborn, B. 1995a. *In vitro*, cleavage and joining at the viral origin of replication by the replication initiator protein of *Tomato yellow leaf curl virus*. *Proceeding of National Academy of Sciences*. USA. 92: 3879-3883.

Lucas, W. J. 2006. Plant viral movement proteins: agents for cell-to-cell trafficking of viral genomes. *Virology* 344:169-184.

Lucas, W.J. 1995. Plasmodesmata: intercellular channels for macromolecular transport in plants. *Curr. Opin. Cell Biol.* 7, 673-680.

Mansoor, S., Amin, I., Hussain, M., Zafar, Y., Bull, S., Briddon, R.W. & Markham P.G. 2001. Association of a disease complex involving a begomovirus, DNA1 and a distinct DNA beta with leaf curl disease of okra in Pakistan. Plant Dis. 85: 922.

Mansoor, S., Briddon, R.W., Bull, S.E., Bedford, I.D., Bashir, A., Hussain, M., Saeed, M., Zafar, Y., Malik, K.A., Fauquet, C and Markham, P.G. 2003. Cotton leaf curl disease is associated with multiple monopartite begomoviruses supported by single DNA β. *Archives of Virology* 148: 1969-9186.

Mansoor, S., Khan, S.H., Bashir, A., Saeed, M., Zafar, Y., Malik, K.A., Briddon, R.W., Stanley, J. and Markham, P.G. 1999. Identification of a novel circular single-stranded

DNA associated with cotton leaf curl disease in Pakistan. *Virology* 259: 190-199.

Mansoor, S., Khan, S.H., Hussain, M., Zafar, Y. Pinner, M.S., Briddon, R.W., Stanley, J. and Markham, P.G. 2000b). Association of a begomovirus and nanovirus like molecule with ageratum yellow vein disease in Pakistan. *Plant Disease* 84: 101.

Mubin, M., Briddon, R.W. and Mansoor, S. 2009b. Complete nucleotide sequence of chilli leaf curl virus and its associated satellites naturally infecting potato in Pakistan. *Archives of Virology* 154: 365-368.

Murant, A.F. and Mayo, M.A. 1982. Satellites of plant viruse. *Annual Review of Phytopathology* 20: 49-70.

Navot, N., Pichersky, E. Zeiden, M. and Czosnek, H. 1991. Tomato yellow leaf curl virus: a whitefly transmitted geminiviruses with a single genomic molecule. *Virology* 185: 151-161.

Noueiry, A.O., Lucas, W.J. and Gilbertson, R.L. 1994. Two protein of a plant DNA virus coordinate nuclear and plasmodesmatal transport. *Cell* 76: 925-932.

Orozco, B.M. and Hanley-Bowdoin, L. 1998. Conserved sequence and structural motifs contribute to the DNA binding cleavage activities of a geminivirus replication protein. *Journal of Biological Chemistry* 273: 24448-24456.

Orozco, B.M., Miller, A.B., Stellage, S.B. and Hanley-Bowdoin, L. 1997. Functional domains of a geminivirus replication protein. *Journal of Biological Chemistry* 272:9840-9846.

Padidam, M., Beachy, R.N. and Fauquet, C.M. 1996. The role of AV2 (pre-coat) and coat protein in viral replication and movement in tomato leaf curl geminivirus. *Virology* 224: 390-404.

Padidam, M., Beachy, R.N., and Fauquet, C.M. 1995b. Classification and identification of geminiviruses using sequence comparisons. *Journal of General Virology* 76: 249-263.

Pandey, P., Choudhury, N.R. and Mukherjee, S.K. 2009. A geminiviral amplicon (VA) derived from *Tomato leaf curl virus* (ToLCV) can replicate in a wide variety of plant species and also acts as a VIGS vector. *Virology Journal* 6: 152.

Pant, V., Gupta, D., Choudhury, N.R., Malathi, V.G., Varma, A. and Mukherjee, S.K. 2001. Molecular characterisation of the Rep protein of the blackgram isolate of Indian mungbean yellow mosaic virus. *Journal of General Virology* 82: 2559-2567.

Paprotka, T., Metzler, V. and Jeske, H. 2010. The first DNA 1-like α satellite in association with New World begomoviruses in natural infections. *Virology* 404: 148-157.

Pascal, E., Sanderfoat, A.A., Ward, B.M., Medville, R., Turgeon, R. and Lazarowitz, S.G. 1994. The geminivirus BR1 movement protein bind ssDNA and localizes to the cell nucleus. *Plant Cell* 6: 995-1006.

Pascle, E.,Goodlove, P.E., Wu, L.C. and Lazarowitz, S.G. 1993. Transgenic plants expressing the BL1 protein exhibit symptoms of viral disease. *Plant Cell* 5: 795-807.

Patil, B.L. and Fauquet, C.M. 2010. Differential interaction between cassava mosaic geminiviruses and geminivirus satellites. *Journal of General Virology* 91: 1871-1882.

Pooma, W. and Petty, I.T.D. 1996. Tomato golden mosaic virus ORF AL4 is genetically distinct from its C4 analogue in monopartite geminivirus. *Journal of General Virology* 77: 1947-1951.

Preiss, W. and Jeske, H. 2003. Multitasking in replication is common among geminiviruses. *Journal of Virology* 77: 2972-2980.

Reddy, C.R.V., Colvin, J., Muniyappa, V. and Seal, S. 2005. Diversity and distribution of begomoviruses infecting tomato in India. *Archives of Virology* 150: 845-867.

Ridgen, J.E., Dry, I.B., Mullineaux, P.M. and Rezian, M.A. 1993. Mutagenesis of the virion sense open reading frames of tomato leaf curl geminiviruses. *Virology* 193: 1001-1005.

Ridgen, J.E., Krake, L.R., Rezian, M.A. and Dry, I.B. 1994. ORF C4 of tomato leaf curl geminivirus is a determinant of symptoms severity. *Virology* 204: 847-850.

Rogers, S.G., Bisaro, D.M., Horsch, R.B., Fraley, R.T., Hoffman, N.L., Brand, L., Elmer, J.S. and Lloyd, A.M. 1986. Tomato golden mosaic virus a component DNA replicates autonomously in transgenic plants. *Cell* 45: 593-600.

Rojas, M. R., Jiang, H., Salathi, R., Xoconostle-Cazares, B., Sundarshana, M.R., Lucas, W.J. and Gilbertson, R.L. 2001. Functional analysis of protein involved in movement of the monopartite begomovirus. Tomato yellow leaf curl virus, *Virology* 291:110-125.

Rojas, M.R., Noueiry Ao, Lucas WJ and Gilbertson, R.L 1998. Bean dwarf mosaic geminivirus movement proteins recognize DNA in a form-and size-spacing manner. *Cell* 95: 105-113.

Romay, G., Chirinos, D., Geraud-Pouey, F. and Desbiez, C. 2010. Association of an atypical alphasatellite with a bipartite New World begomovirus. *Archives of Virology* 55, 1843-1847.

Rouhibakhsh, A. and Malathi, V.G. 2005. A severe leaf crinkle disease of cowpea caused by *Mungbean yellow mosaic India virus* and a satellite DNA β. *Plant Pathology* 54: 259.

Rybicki, E.P. 1994. A phylogenetic and evolutionary justification for three genera geminiviridae. *Archives of Virology* 139: 49-78.

Sanderfoot, A.A. and Lazarowitz, S.G. 1995. Cooperation in viral movement: the geminivirus BL1 movement protein interacts with BR1 and redirects if from the nucleus to the cell periphery. *Plant Cell* 7: 1185-1194.

Sanderfoot, A.A., Ingham, D.J. and lazarowitz, S.G. 1996. A viral movement protein as a nuclear shuttle. The geminivirus BV1 movement protein contains domains essential for interaction with BC1 and nuclear localization. *Plant Physiology* 110: 23-33.

Saunders, K. & Stanley J. 1999. A nanovirus-like component associated with yellow vein disease of *Ageratum conyzoides*: evidence for inter-family recombination between plant DNA viruses. *Virology* 264: 142-152.

Saunders, K. and Stanley, J. 1995. Complementation of African cassava mosaic virus AC2 gene function in a mixed bipartite geminivirus infection. *Journal of General Virology* 76: 2287-2292.

Saunders, K., Bedford, I.D., Briddon, R.W., Markham, P.G., Wong, S.M., Stanley, J. 2000. A unique virus complex causes Ageratum yellow vein disease. *Proceeding of National Academy of Science of the United Stated of America* 97: 6890-6895.

Saunders, K., Lucy, A. and Stanley, J. 1992. RNA-primed complemantry-sense DNA synthesis of the geminivirus: African cassava mosaic virus. *Nucleic Acids Research* 20: 6311-6315.

Settlage, S.B., Miller, A.B. and Hanley-Bowdoin, L. 1996. Interaction between geminivirus replication proteins. *Journal of Virology* 70: 6790-6795.

Settlage, S.B., Miller, A.B., Grussem, W. and Hanley-Bowdoin, L. 2001. Dual interaction of a geminivirus replication accessory factor with a viral replication protein and a plant cell cycle regulator. *Virology* 279: 570-576.

Sharma, P. and Ikegami, M. 2009. Characterization of signals that dictate nuclear/nucleolar and cytoplasmic shuttling of the capsid protein of *Tomato leaf curl Java virus* associated with DNAβ satellite. *Virus* Research 144: 145-153.

Snehi, S. K., Khan, M. S., Raj, S. K., and Prasad, V. 2011a. Complete nucleotide sequence of Croton yellow vein mosaic virus and DNA-β associated with yellow vein mosaic disease of *Jatropha gossypifolia* in India, *Virus Genes* 43: 93-101.

Srivastava, A., Raj, S. K., Kumar, S. and Snehi, S. K. 2013a. New record of *Papaya leaf curl virus* and Ageratum leaf curl beta satellite associated with yellow vein disease of aster in India. *New Disease Reports* 28, 6.

Srivastava A., Raj S.K., S. Kumar, S. K. Snehi, Kulshreshtha A., Hallan V. and Pande S. S. 2013b. Molecular identification of Ageratum enation virus, betasatellite and alphasatellite molecules isolated from yellow vein diseased Amaranthus cruentus in India. *Virus Genes* 47 (3): 584-590.

Stanley, J. 1985. The molecular biology of geminiviruses. *Advances in Virus Research* 30: 139-177.

Stanley, J. 1995. Analysis of African cassava mosaic virus recombinants suggests strand nicking occurs within the conserved nonanucleotide motif during the initiation of rolling circle DNA replication. *Virology* 206: 707-712.

Stenger, D.C., Revington, G.N., Stevenson, M.C. and Bisaro, D.M. 1991. Replication release of geminivirus genomes from randomly repeated copies: evidence for rolling circle replication of a plant viral DNA. *Proc. Natl. Acad. Sci.* USA. 88: 8029-8033.

Sudarshana, M.R., Wang, H.L., Lucas, W.J. and Gilbertson, R.L. 1998. Dynamics of bean dwarf mosaic geminivirus cell-to-cell and long-distance movement in *Phaseolus vulgaris* revealed, using the green fluorescent protein. *Molecular Plant–Microbe Interactions* 11: 277-291.

Sung, Y.K. and Coutts, R.H.A. 1995. Mutational analysis of *Potato yellow mosaic geminivirus*. *Journal of General Virology* 76: 1773-80.

Sunter, G. and Bisaro, D.M. 1989. Transcription map of the B genome component of tomato golden mosaic virus and comparison with A component transcripts. *Virology* 173: 647-655.

Sunter, G. and Bisaro, D.M. 1991. Transactivation in a geminivirus AL2 gene product is needed for coat protein expression. *Virology* 180: 416-419.

Sunter, G. and Bisaro, D.M. 1992. Transactivation of geminivirus AR1 and BR1 gene expression by the viral AL2 gene product occurs at the level of transcription. *Plant Cell*. 4: 1321-1331.

Sunter, G. and Bisaro, D.M. 1997. Regulation of a geminivirus coat protein promoter by AL2 protein (TrAP): Evidence for activation and derepression mechanism. *Virology* 232: 269-280.

Sunter, G., Gardiner, W., E., Rushing, A., E., Rogers, S. G. and Bisaro, D. M. 1987. Independent encapsidation of tomato golden mosaic virus A component DNA in transgenic plant. *Plant Mol. Biol.* 8: 477-484.

Sunter, G., Hartitz, M.D. and Bisaro, D.M. 1993. Tomato golden mosaic leftward gene expression: autoregulation of geminivirus replication protein. *Virology* 195: 275-280.

Sunter, G., Hartitz, M.D., Hormuzdi, S.G., Brough, C.L. and Bisaro, D.M. 1990. Genetic analysis of tomato golden mosaic virus. ORF AL2 is required for coat protein accumulation while ORF AL3 is necessary for efficient DNA replication. *Virology* 179: 69-77.

Sunter, G., Hartitz, M.D., Hormuzdi, S.G., Brough, C.L. and Bisaro, D.M. 1990. Genetic analysis of tomato golden mosaic virus. ORF AL2 is required for coat protein accumulation while ORF AL3 is necessary for efficient DNA replication. *Virology* 179: 69-77.

Sunter, G., Stenger, D.C. and Bisaro, D.M. 1994 Heterologous complementation by geminivirus AL2 and AL3 gene. *Virology* 203:203-210.

Tahir, M. and Haider, M.S. 2005. First report of *Tomato leaf curl New Delhi virus* infecting bitter gourd in Pakistan. *Plant Pathology* 54: 807

Tiendrébéogo, F., Lefeuvre, P., Hoareau, M., Villemot, J., Konate, G., Traoré, A.S., Barro, N., Traoré, V.S, Reynaud, B., Traoré, O. and Lett, J.M. 2010. Molecular diversity of *Cotton leaf curl Gezira virus* isolates and their satellite DNAs associated with okra leaf curl disease in Burkina Faso. *Virology Journal* 7: 48.

Townsend, R., watts, J., and Stanley, J. 1986. Synthesis of viral DNA form in *Nicotiana plumbaginifolia* protoplast inoculated with cassava latent virus (CLV): evidence for the independent replication of one component of CLV genome. *Nucleic Acid Research* 14: 1253-1265.

Trinks, D., Rajeswaran, R., Shivaprasad, P.V., Akbergenov, R., Oakeley, E.J., Veluthambi, K., Hohn, T. and Pooggin, M. 2005. Suppression of RNA silencing by a geminivirus nuclear protein, AC2, correlates with transactivation of host genes. *Journal of Virology* 79: 2517- 2527.

van Wezel, R., Dong X., Blake P., Stanley J., and Hong Y. 2002a. Differential roles of geminivirus Rep and AC4 (C4) in the induction of necrosis in *Nicotiana benthamiana*. *Molecular Plant Pathology* 3: 461-471.

van Wezel, R., Dong X., Liu, H., Tien D., Stanley J., and Hong Y. 2002b. Mutations in three cytosine residues in tomato yellow leaf curl virus China C2 protein causes dysfunction in pathogenesis and post transcriptional and gene silencing suppression. *Mol. Plant microbe Interact.* 15: 203-208.

van Wezel, R., Liu, H., Tien P., Stanley J., and Hong Y. 2001. Gene C2 of the monopartite geminiviruses tomato yellow leaf curl virus -China encodes a pathogenicity determinant that is localized in the nucleus. *Molecular Plant microbe-Interact* 14: 1125-1128.

Vanitharani, R., Chellappan, P., Pita, J.S. and Fauquet, C.M. 2004. Differential roles of AC2 and AC4 of cassava geminiviruses in mediating synergism and suppression of posttranscriptional gene silencing. *Journal of Virology* 78: 9487-9498.

Varma, A. and Malathi, V.G. 2003. Emerging geminivirus problems: A serious threat to crop production. *Annals of Applied Biology* 142: 145-146.

Voinnet, O., Pinto, Y.M. and Baulcombe, D.C. 1999. Suppressor of gene silencing: a general strategy used by diverse DNA and RNA viruses of plants. *Proc. Natl. Acad. Sci.* USA 96: 14147-14152.

von Arnim, A., Frischmuth, T. and Stanley, J. 1993. Detection and possible functions of ACMV DNA-B gene products. *Virology* 192: 264-272.

von Arnin, A. and Stanley , J. 1992. Inhibition of *African cassava mosaic* systemic infection by a movement protein from the related geminivirus *Tomato golden mosaic virus*. *Virology* 187: 555-564.

Wang, H., Hao, L., Shung, C.Y., Sunter, G. and Bisaro, D.M. 2003. Adenosine kinase is inactivated by geminivirus AL2 and L2 proteins. *Plant Cell* 15: 3020-3032.

Wartig, L., Kheyr-pour, A., Noris, E., Kouchkovsky, F.D., Jouanneau, F., Gronenborn, B. and Jupin, I. 1997. Genetic analysis of the monopartite Tomato yellow leaf curl geminivirus: Roles of V1, V2 and C2 ORFs in viral pathogenesis. *Virology* 228: 132- 140.

Xie, Y., Wu, P., Liu, P., Gong, H. and Zhou., X. 2010. Characterization of alphasatellites associated with monopartite begomovirus/betasatellite complexes in Yunnan, China. *Virology Journal* 7: 178.

Xiong, Q., Guo, X.j., Che, H.Y. and Zhou, X.P. 2005. Molecular characterization of a distinct begomovirus and its associated satellite DNA molecule infecting *Sida acuta* in

China. *Journal of Phytopathology* 153: 264-268.

Yadava, P., Suyal., G. and Mukherjee, S.K. 2010. Begomovirus DNA replication and pathogenicity. *Current Science* 98: 360-368.

Yang, J-Y., Iwasaki, M., Machinda, C., Machinda, Y., Zhou, X. and Chua, N-H. 2008. βC1, the pathogenicity factor of TYLCCNV, interacts with AS1 to alter leaf development and suppress selective jasmonic acid responses. *Genes Development* 22: 2564-2577.

Zhan, X.C., Haley, A., Richardson, K. and Morris, B. 1991. Analysis of the potential promoter sequences of African cassava mosaic virus by transient expression of the β-glucuronidase gene. *Journal of General Virology* 72: 2849-2852.

Zhang W., Olson N.H., Baker T.S., Paulkner L., Agbandje Mc Kenna M., Baulton M.I., Devis J.W., and Mekenna R. 2001. Structure of the maize streak virus geminate particle. *Virology* 279: 471-477.

Zhou, X., Xie, Y., Tao, X., Zhang, Z., Li, Z. and Fauquet, C.M. 2003. Characterization of DNA β associated with begomoviruses in China and evidence for co-evolution with their cognate viral DNA-A. *Journal of General Virology* 84: 237-247.

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