Biological Times

Volume No. 5 | Issue No. 2 | Pages 21-23

Genomic Plasticity and Molecular Evolution of Begomoviruses

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ABSTRACT

The most economically significant and largest family of plant infectious ssDNA viruses is Geminiviridae, which causes devastating diseases in cotton, tomato, cassava and papaya crops throughout the world. Begomoviruses are very diverse (they have small genomes with ~2.6-2.8 kb in length) and evolve at high rates with high rates of mutation, recombination, and pseudorecombination, as well as interactions with betasatellites and alphasatellites. Recombination hotspots at the intergenic region, replication-associated protein and coat protein gene tend to result in epidemic strains, whereas the satellite associations tend to increase pathogenicity and adaptability. Recent developments in the high-throughput sequencing and molecular phylogenetics have shown the intricate evolution patterns, such as expansion of host range and geographic organization. Awareness of these molecular processes is critical in forecasting the viral outbreaks, developing long-term crops that develop resistance, and enhance global food security and agricultural sustainability through enhancing protection measures of crops.

Keywords: Genomic plasticity, molecular evolution, recombination, host adaptation, ssDNA viruses

To cite this article: Yasmeen E, B Ghaffar, Z Bibi, N Khan, A Maqsood & MY Malik. Genomic Plasticity and Molecular Evolution of Begomoviruses. Biological Times. 2026. February 5(2): 21-23.

Introduction

Begomoviruses represent the most varied genus of the *Geminiviridae* family, being circulated by the whitefly (*Bemisia tabaci*) in a persistent form (1). They cause numerous species of dicotyledon plants and devastating diseases that have devastating economic effects, including cotton leaf curl disease, tomato yellow leaf curl disease and cassava mosaic disease (2-5). In extreme epidemic situations, losses in yields due to begomoviruses can be as high as 100% in the tropical and subtropical regions, which is a significant threat to food security and agro sustainability (Table 1) (6). The worldwide spread of the cryptic species of *B. tabaci*, augmented global commerce of plant materials, monocropping, and climate change have all augmented the onset, adjustment and geographic diffusion of new begomovirus variants (7).

Table 1: Major Crops Affected by Begomoviruses and Representative Species

Crop	Representative Begomovirus Species	Geographic Distribution	Economic Impact
Cotton	Cotton leaf curl virus complex	South Asia, Africa	Severe yield loss
Tomato	Tomato yellow leaf curl virus	Worldwide	Major horticultural losses
Cassava	Cassava mosaic begomoviruses	Africa	Food security threat
Papaya	Papaya leaf curl virus	Asia	Reduced fruit yield

Begomoviruses have circular single-stranded DNA (ssDNA) genomes contained in typical icosahedral twinned particles (8). They can have monopartite or bipartite genome with either one DNA-A-like component or DNA-A and DNA-B components of about 2.6-2.8 kb each (9). In spite of their small genome size, begomoviruses have remarkable species and strain genetic variability. This seemingly contradictory small genome but massive diversity indicates high levels of genomic plasticity which is a characteristic trait that forms the foundation of their success as an ecological force and an evolutionary stronghold (10).

The high substitution rates, frequent recombination, pseudorecombination of components of the genome, and dynamic interactions with satellite DNAs are the major factors that lead to genomic plasticity in begomoviruses (11-8). The recombination especially has been known to be a significant force in the evolution with the creation of chimeric genomes that differ in host range, virulence and epidemiological features (12-13). Molecular evolutionary studies also indicate that vital genes tend to be highly purifying selected, whereas host switch and adaptations of vectors are followed by episodic positive selection (14).

This review evaluates the genomic architecture of begomoviruses and synthesizes the existing knowledge about the mechanisms of evolution and diversification of begomoviruses and focuses on recombination dynamics, satellites-mediated modulation, and adaptive processes which allow the begomoviruses to expand their host and epidemics.

Genome Organization and Functional Architecture

Old World and New World Begomoviruses belong to distinct lineages, which represent a geographic isolation and evolutionary splits (1). Old World begomoviruses are common monopartite genomes commonly linked to betasatellites whereas New World begomoviruses are mostly bipartite and usually have no satellites (15).

DNA-A Component

The DNA-A constituent is the indispensable replicative element of begomoviruses and it encompasses the proteins that are needed in viral replication, regulation of transcription and encapsidation. The DNA-A strand, complementary sense, codes replication-associated protein Rep/AC1, transcriptional activator protein TrAP/AC2, replication enhancer protein REn/AC3 and C4 protein (9). The key activator of rolling-circular replication (RCR) is Rep, which inserts a site specific nick into the conserved nonanucleotide sequence (TAATATT↓AC) in the intergenic region (IR) (16). Such interactions between Rep and iteron sequences that are flanks of the place of replication cause recruitment of host DNA polymerases and related replication factors, and thus coordination of viral genome amplification. In addition to replication, Rep is also involved in the control of cell cycles and interaction between the hosts, which allows the virus to establish a replication-permissive state of differentiated plant cells (17).

TrAP is a transcriptional regulator, which activates the expression of the virion-sense genes and suppresses host RNA silencing defenses whereas REn promotes replication efficiency by interacting with Rep and host factors. C4 protein has been identified to mediate in symptom development, host signaling pathway modulation and interference to defense responses. DNA-A on the virion-sense strand encode the coat protein (CP/AV1) required in encapsidation of viral ssDNA, transmission by vectors on *B. tabaci*, and nuclear trafficking of viral genomes. Host range specificity and the adaptation of vectors have been identified to be variability within CP (18).

DNA-B Component

The DNA-B part of bipartite begomoviruses contains two proteins associated with movement; the movement protein (MP/BV1) and the nuclear shuttle protein (NSP/BC1). NSP helps in the movement of viral DNA in the nucleus to the cytoplasm, and MP assists in the transmission of cells between each other, via the plasmodesmata, facilitating infection at the systemic level (15). Specific interactions between Rep and iteron motifs in the IR determine functionality compatibility between DNA-A and DNA-B components largely, as both components have to be replicated in a coordinated way.

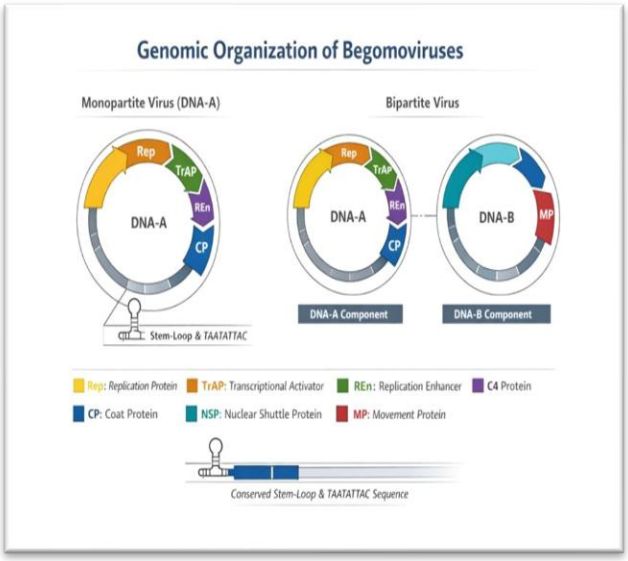

Fig. 1: Schematic representation of monopartite and bipartite begomovirus genome organization showing major open reading frames and the conserved stem-loop region.

Modular Genome Structure

There is a significant functional modularity in the genome of the begomovirus, and replication, movement, encapsidation and pathogenicity determinants being divided into separate but exchangeable regions. This enabled a modular architecture that allows it to recombine and to have different components without necessarily affecting key biological processes. This allows genetic mixing with close relatives of viruses, resulting into viable recombinant offspring, which has a great contribution to genomic plasticity and evolutionary diversification (13).

Mechanisms Driving Genomic Plasticity

Mutation and Substitution Rates

Begomoviruses are remarkable since they are dependent on host DNA polymerases to replicate their genome but the rate of mutation and substitution is unexpectedly high which is close to that of RNA viruses (10). There are a number of reasons that cause this high evolution rate. The errors of the rolling-circle replication process mediated by the viral Rep protein can occur when the strand is being displaced, especially when the host polymerase is infidel. Besides, viral ssDNA oxidative damage during infection and intracellular replication can also lead to mutations that accumulate with the subsequent replication cycles (19).

Population genetic studies have continued to indicate that key genes of the virus, such as the replication related protein (Rep) and coat protein (CP) have experienced intense purifying selection, meaning that the majority of amino acid modifications are damaging and eliminated by natural selection (14). This limitation maintains vital functions such as initiation of replication, encapsidation and host/vector interactions. However, local positive selection has been observed in particular locations in the CP and Rep genes which can be associated with host adaptation or changed the efficiency of vectors in transmission. Such adaptive changes could give the virus the ability to use new hosts, or maximize interactions with *B. tabaci* vectors, leading to the spread of epidemics (20).

Recombination as one of the key forces of evolution

Mutation adds small changes, whereas recombination is a significant contributor towards begomovirus diversification. Intra- and inter-species recombination is common and more so in mixed infections where more than one begomovirus infects a given host plant (11,13). The recombination breakpoints are usually identified in intergenic region (IR), Rep and CP genes (13). The IR is especially recombination-prone because it has structures of origin-of-replication as well as secondary motifs that allow strand exchange. Epidemic strains such as those that cause cotton leaf curl disease and tomato yellow leaf curl disease have been formed as a result of recombination, and as such, it has become highly important in the emergence and epidemiology of viruses (12). Phylogenetic incongruence of various genomic regions, e.g. the different tree topologies of CP and Rep, is a powerful molecular support of recombination and genome mosaic.

Pseudorecombination and Genome Component Exchange

The re-assortment of heterologous DNA-A and DNA-B components is called pseudorecombination in bipartite begomoviruses. The experimental works show that viable pseudo recombinants may be formed in case Rep-iteron compatibility is maintained and both parts may replicate in coordination (21). These interactions have the capability of changing host range, severity of symptoms and geographic adaptability and contribute to the development of new epidemic strains.

Evolutionary Flexibility and Satellite Molecules

Old World begomoviruses tend to co-exist with betasatellites (1.3 kb) and alphasatellites, which increase viral plasticity, further (8). Betasatellites encode one βC1 protein that is a pathogenicity determinant and RNA silencing suppressor, which exacerbates disease symptoms and allows adaptation to novel hosts (22). Rep-like proteins are encoded in the alphasatellites and can autonomously replicate, although they are encapsidated by a helper begomovirus in order to be transferred throughout the body. These interactions of satellites substantially broaden phenotypic and evolutionary diversity of begomovirus complexes, which offers further means of rapid adaptation and epidemic outbreaks.

Host Adaptation and Molecular Evolution

Host range expansion is an example of a characteristic property of begomovir evolution and a foundation of the ecological success and worldwide agricultural effect of begomoviruses. Begomoviruses have a wide range of dicotyledonous hosts and changes in host often relate to adaptive genetic alterations. CP, the protein expressed by AV1 gene in monopartite viruses and DNA-A in bio-partite viruses, is a vital factor in determining the specificity of vectors, compatibility with the host and dissemination throughout the body. Since the CP affects the contacts with the whitefly *B. tabaci* and host cellular factors, there are subtle amino acid changes that may impact transmission capability and infection processes. CP adaptive changes have been associated with increased transmission of vectors, and increased host range, which is significant in the determination of viral fitness (3).

Even with the very high adaptability, selective pressure studies of genomes of begomoviruses are always able to show that the majority of the coding regions are under strong purifying (negative) selection. This trend is attributed to the functional limitations placed on them by their small genomes, overlapping open reading frames, and multifunctional proteins. Groundbreaking genes, including Rep, CP and movement proteins, need to have structural and functional integrity, which restricts the acquisition of destructive mutations. Episodic positive selection has however been observed at particular codons sites especially when there is a change of host or during adaptation to new ecological niche or in case of an epidemic boom (14). Such flashes of adaptive evolution can promote avoidance of host defense or optimization of virus -vectors interactions.

The recent appearance of cotton leaf curl disease (CLCuD) complexes gives a good example of host adaptation through recombination and epidemic outbreak. In the example, the interspecies recombination among begomoviruses that was accompanied by the acquisition and evolution of betasatellites that encode pathogenicity determinants like 2C1 in this case helped to break through the resistance and resulted in massive outbreaks in cotton-producing areas (23-25). This depicts the manner in which genomic plasticity, in combined with selection pressures, spurs host range expansion and the development of disease.

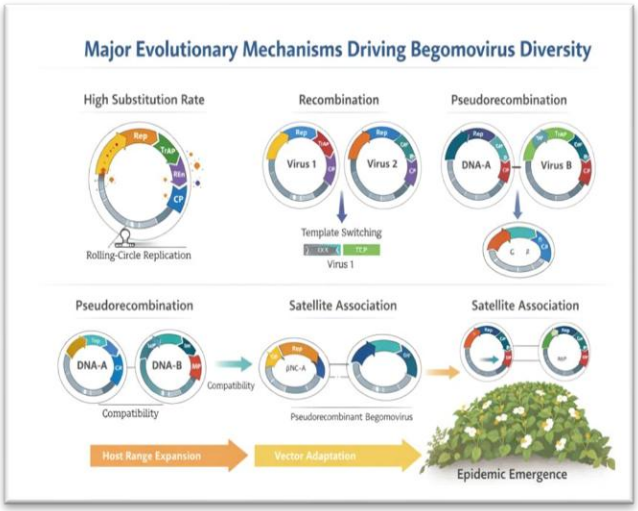

Fig. 2: Major evolutionary mechanisms contributing to begomovirus diversity, including mutation, recombination, pseudorecombination, and satellite interaction.

Phylogenomic Insights into Diversification

Extensive phylogenetic studies on full genome sequences have continuously demonstrated the presence of two primary evolutionary groups of begomoviruses, which are Old World (OW) and New World (NW) clades, which are indicative of historic geographic separation and independent evolutionary development (1, 23, 27). Old World

begomoviruses (common in Africa, Europe, Asia and Australasia) are most commonly monopartite and commonly in association with betasatellites and alphasatellites. New world begomoviruses, which are mostly restricted to the Americas, are in contrast to a large degree bipartite and usually non-satellite. Such a phylogenetic dichotomy implies historical biogeographic barriers and divergences in evolutionary pressures that condition the organization of the genome and patterns of diversification of lineages (23-27).

High throughput sequencing (HTS) technologies have immensely improved our knowledge of the begomovirus diversity. Metagenomic surveys have revealed a great deal of cryptic variation, such as low-frequency variants, previously unsuspected species, and complicated mixed infections in individual host plants (27). It is especially important that mixed infections bring about the possibilities of genetic exchange due to recombination and reassortment, which promotes the production of new genotypes faster (24, 26-27). Phylogenomic studies can also show mosaic genome patterns in the begomoviruses that can be interpreted as evidence of repeated interspecies recombination events, highlighting the dynamic nature of evolutionary topography of the begomoviruses.

The phylogeography research also shows that human activities like international trade in flora resources, agricultural expansion, and worldwide transportation of whitefly vectors have supported extensive dissemination of begomoviruses (23). Climate change, which affects the dynamics of vectors populations and increases the area of their proper habitat, is also a cause of changes in the distribution of viruses and the occurrence of epidemics in the areas that have not been affected previously. This means that an inherent set of molecular processes including mutation and recombination is acted on by environmental and ecological factors that interact in a complex manner to define the trends of diversification and global dissemination (25).

Prospects, Management of the disease, and conclusion

Comprehensive interpretation of the begomovirus genomic plasticity is an essential feature in the development of sustainable and sound management plans of disease control. The begomoviruses have frequently subverted established control mechanisms, such as vector control and traditional resistance breeding that is attributed to the high evolutionary rates. These viruses can be used to beat the host resistance genes through evolution of new recombinant strains, high mutation rates, and frequent recombination through a relatively short period of time. Consequently, the resistant methods should take into consideration the evolutionary possibilities of the begomovirus populations.

Recent developments in genome editing methods and specifically the CRISPR/Cas systems have presented an opportunity into antiviral resistance where the target of the intervention is an evolutionarily preserved viral genome (Rep) or intergenic region (27). CRISPR-mediated interference has the ability to attenuate viral accumulation and symptom development to a large extent. Nevertheless, the begomoviruses have a high recombination rate and adaptive ability that poses the risk of the occurrence of escape mutants with modified target sequences. This underscores the importance of multiplexed targeting strategies to be able to cause degradation of various conserved viral regions at the same time to reduce resistance decomposition.

Along with molecular resistance measures, integrative management is also required in long-term management. They are genomic surveillance to track viral diversity and new recombinants, evolutionary modeling to forecast the risks of outbreaks and implementation of resistant cultivar based on population genome information. The molecular level of interaction between virus, vectors and hosts will also help in the advancement of sustained resistance and the establishment of specific intervention approaches.

To sum up, begomoviruses are one example of how small DNA viruses can be enormously successful in evolution due to the heavy reliance on the wonderful plasticity of the genome. Genetic diversification, host adaptation and emergence of epidemics all occur as a result of mutation, recombination, pseudorecombination and satellite associations. Further development of genomics, phylogenetics, and molecular plant pathology will strengthen our capacity to predict evolutionary patterns and reduce crop losses caused by begomoviruses and increase agricultural sustainability in the world.

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