Agritech, March, 2026, Vol.3(1): 12-14

Cassava Mosaic Virus: Mechanisms of Infection, Host Interactions, and Disease Dynamics in Cassava

B. Lohitha[1,2], T. Makeshkumar[1], K. Krishnaveni[1,2], S. Divya[1,3], S. Pavitra[1,4]

[1]ICAR- Central Tuber Crops Research Institute, Thiruvanathapuram, Kerala
[2]ICAR-IARI-Indian Institute of Horticultural Research, Bengaluru, Karnataka
[3]ICAR-Indian Agricultural Research Institute, New Delhi, Delhi
[4]University of Kerala, Kariavattom, Thiruvanathapuram, Kerala

makeshctcri@gmail.com

Cassava (*Manihot esculenta*) is a staple and industrial crop cultivated for its starchy storage roots, providing caloric security for more than 800 million people worldwide. However, its production is severely hampered by viral diseases, particularly Cassava Mosaic Disease (CMD). CMD is characterized by leaf chlorosis, mosaic patterning, stunting, and reduced root yield, often leading to total crop failure in severe outbreaks. CMD is caused by a complex of single-stranded DNA viruses classified within the genus Begomovirus (family Geminiviridae). These geminiviruses possess circular, bipartite genomes and are transmitted primarily by the whitefly vector Bemisia tabaci. The complexity of CMD arises not only from the diverse viruses involved but also from their capacity to recombine, adapt, and effectively subvert host plant defences (Hareesh et al., 2023).

Viral Structure and Genome Organization

Cassava mosaic viruses are bipartite begomoviruses with two circular single-stranded DNA components, DNA-A and DNA-B. The DNA-A component encodes proteins essential for viral replication, gene regulation, encapsidation, and suppression of host defense responses. In DNA-A, ***AV1*** encodes the coat protein required for particle formation and vector transmission, while ***AV2*** contributes to virus movement and pathogenicity. The complementary-sense genes include ***AC1***, which encodes the replication-associated protein initiating viral DNA replication, ***AC2*** (transcriptional activator protein) involved in viral gene expression and silencing suppression, ***AC3*** (replication enhancer protein) that enhances replication efficiency, and ***AC4***, which plays a role in symptom development and RNA silencing suppression. The DNA-B component encodes movement-related proteins, where ***BV1*** functions as the nuclear shuttle protein and ***BC1*** acts as the movement protein enabling cell-to-cell spread. Both genome components share a conserved ***common region (CR)*** containing the origin of replication with a characteristic stem-loop structure. The intergenic regions, including the long intergenic region (LIR) and short intergenic region (SIR), regulate transcription and replication. Together, DNA-A and DNA-B coordinate replication and systemic movement, leading to the characteristic symptoms of cassava mosaic disease (Pati & Fauquet, 2009).

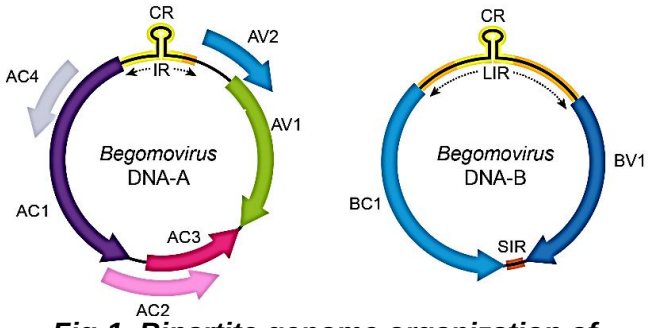

Fig-1. Bipartite genome organization of cassava mosaic virus (Begomovirus) showing DNA-A and DNA-B components

Transmission and Early Infection Dynamics

CMD viruses are transmitted in the field primarily through a persistent, circulative, non-propagative manner by whiteflies (*Bemisia tabaci*). Adult whiteflies acquire the virus when feeding on infected plants; the virus circulates through the midgut and reaches the salivary glands, from where it can be transmitted to new host plants during feeding. Apart from vector transmission, CMD is also perpetuated through the use of infected stem cuttings a common propagation method in cassava cultivation which leads to vertical transmission of the virus across planting cycles. Upon entering cassava tissues, CMGs localize to young leaves and actively dividing cells, establishing infection in mesophyll and phloem components. The viral ssDNA is rapidly converted to a double-stranded replicative form by host DNA polymerases, setting the stage for active replication and gene transcription (Al Basir et al., 2021).

Host–Virus Molecular Interactions

Hijacking Host Replication

Cassava mosaic viruses lack their own DNA replication machinery; instead, they rely entirely on host nuclear replication systems. The begomovirus Rep protein initiates replication via a rolling-circle mechanism by binding to the conserved stem-loop structure in the intergenic region and recruiting host DNA replication proteins. The viral double-stranded DNA associates with host histones, forming minichromosomes that facilitate the transcription of viral genes by host RNA polymerase II. This integration of viral DNA into host chromatin architecture allows the virus to evade early detection and exploit cellular transcription mechanisms.

Manipulation of the Cell Cycle

One of the most critical aspects of CMD pathogenesis is the ability of CMGs to manipulate the host cell cycle. Rep interacts with host cell-cycle regulators, disrupting the retinoblastoma (Rb)-E2F pathway and forcing differentiated leaf cells back into a replication-competent state. This reactivation creates a cellular environment with abundant nucleotides and replication factors that benefit viral genome amplification, but at the expense of normal cell differentiation and growth.

Suppression of Host Defence: RNA Silencing

Plants defend against viruses through RNA silencing mechanisms involving small interfering RNAs (siRNAs). However, CMGs have evolved viral suppressors of RNA silencing (VSRs) that interfere with siRNA biogenesis and function. These suppressors bind to siRNAs or interact with components of the RNA-induced silencing complex (RISC), preventing the degradation of viral nucleic acids. By suppressing host RNA silencing, CMGs effectively disarm a primary antiviral defense system, allowing widespread viral replication.

Metabolic Disruption and Symptom Expression

In addition to replication and defense suppression, CMGs disrupt host plant metabolism. Proteomic analyses show significant changes in protein expression profiles in infected cassava tissues, including proteins involved in transcriptional regulation, chromatin modification, and stress responses. For example, the AT-hook motif nuclear localized protein AHL22 interacts with histone deacetylases, implicating altered transcriptional control during infection, which may contribute to the gene silencing observed in infected plants. Symptom development in CMD is linked to chloroplast dysfunction and reduced photosynthetic activity. Chlorotic mosaic and yellowing reflect compromised chlorophyll biosynthesis, while leaf distortion arises from disrupted leaf development pathways. These symptomatic manifestations ultimately reduce photosynthate availability for root tuber development, leading to significant yield losses (Siriwan et al., 2022).

Genetic Diversity, Recombination, and Evolution of CMD Viruses

One of the most striking features of cassava mosaic virus evolution is the role of recombination in shaping CMG diversity. Genetic studies indicate that recombination events are pervasive among CMGs, resulting in new species and strains with altered virulence and host adaptation profiles. A comprehensive analysis of full-length DNA-A and DNA-B sequences from recognized CMG species revealed extensive interspecies recombination events, with a majority of species bearing recombination signatures in their history. This dynamic genetic exchange contributes to viral adaptation, emergence of novel virulent forms, and challenges in disease management. Recombination also facilitates the combination of genomic modules from different CMGs, giving rise to viruses with enhanced infectivity or broader host ranges. The observed recombinant origins of many CMG species point to the fundamental role of genetic exchange in viral evolution and speciation (Crespo-Bellido et al., 2021).

Mixed Infections and Synergistic Pathogenesis

Mixed infections, in which a cassava plant is simultaneously infected by more than one CMG, are widespread in endemic regions. Such co-infections can lead to synergistic interactions, resulting in more severe symptoms and enhanced viral replication compared to single infections. Although attempts to form viable recombinants from certain mixed infections in experimental systems have had varied success, the presence of multiple viral components within the same cell provides opportunities for genetic exchange and functional complementation. In some cases, the DNA-A component of one virus may replicate and spread even in the absence of its corresponding DNA-B partner when assisted by another begomovirus, illustrating the complex interplay between co-infecting viruses.

Epidemiological and Ecological Considerations

The dynamics of CMD in the field are influenced by vector biology, host plant susceptibility, and environmental factors.

Mathematical epidemiological models demonstrate how whitefly maturation and infection dynamics influence the spread of CMD at the population level, providing insights into how vector lifecycle events can affect disease incidence. Field surveys show that CMD incidence varies geographically, with certain CMG species dominating particular regions, and patterns of mixed infections shifting

over time due to host and vector pressures. The persistence of infected planting material and the mobility of whiteflies contribute to the sustained propagation of CMD across landscapes.

Host resistance and Breeding Strategies

Plant breeders have identified multiple sources of resistance to CMD in cassava germplasm. Classical resistance loci (such as CMD1, CMD2, and CMD3) confer varying degrees of resistance, though their effectiveness can be compromised by somatic embryogenesis and other factors. Some CMD2-type resistant plants lose resistance after certain tissue culture procedures, underscoring the complexity of host resistance mechanisms. Advances in genetic engineering approaches such as CRISPR-Cas9 offer avenues for developing CMD-resistant cultivars. Gene editing strategies targeting viral replication sequences or host susceptibility factors have shown promise, though the potential for evolution of editing-resistant viral variants underscores the evolutionary arms race between host and pathogen.

Diagnosis and Emerging Technologies

Accurate and rapid diagnosis of cassava mosaic viruses is crucial for effective disease management. High-throughput sequencing technologies such as Oxford Nanopore sequencing improve detection and characterization of viral species and mixed infections directly from plant tissues. These molecular tools enable comprehensive surveillance of CMD populations and help in tracking emergence of new variants.

Impact on Food Security and Socio-Economics

CMD significantly threatens food and economic security in cassava-dependent regions. Yield losses due to CMD can exceed 50% in susceptible cultivars and at times lead to local famines. Historical epidemics have caused declines in cassava production, increased prices, and associated socioeconomic consequences in affected regions.

Integrated Disease Management

Effective CMD management requires an integrated strategy that combines use of virus-free planting material, resistant varieties, whitefly control, and field hygiene practices. Clean seed programs that distribute certified virus-free cuttings, coupled with deployment of resistant cultivars, represent the most sustainable approach to reducing CMD prevalence. Vector management including biological control and cultural practices - complements these efforts (Bhaargavi et al., 2025).

Conclusions and Future Directions

Cassava Mosaic Disease represents a complex biological challenge driven by sophisticated viral strategies that hijack host cellular mechanisms, evade defences, and evolve through recombination and mixed infections. Understanding the molecular basis of host–virus interactions including replication, cell cycle manipulation, defence suppression, and epidemiological dynamics is essential for developing sustainable solutions. Contemporary approaches that integrate molecular breeding, gene editing, advanced diagnostics, and integrated field management hold promise for mitigating the impact of CMD on cassava productivity and food security.

References

- Al Basir, F., Kyrychko, Y. N., Blyuss, K. B., & Ray, S. (2021). Effects of vector maturation time on the dynamics of cassava mosaic disease. Bulletin of mathematical biology, 83(8), 87.
- Bhaargavi, R., Latha, T. K. S., Makeshkumar, T., & Harish, S. (2025). Cassava mosaic disease: strategies for recovery and sustainable management. Australasian Plant Pathology, 54(1), 1-12.
- Crespo-Bellido, A., Hoyer, J. S., Dubey, D., Jeannot, R. B., & Duffy, S. (2021). Interspecies recombination has driven the macroevolution of cassava mosaic begomoviruses. Journal of Virology, 95(17), 10-1128.
- Hareesh, P. S., Resmi, T. R., Sheela, M. N., & Makeshkumar, T. (2023). Cassava mosaic disease in South and Southeast Asia: current status and prospects. Frontiers in Sustainable Food Systems, 7, 1086660.
- Patil, B. L., & Fauquet, C. M. (2009). Cassava mosaic geminiviruses: actual knowledge and perspectives. Molecular plant pathology, 10(5), 685-70
- Siriwan, W., Hemniam, N., Vannatim, N., Malichan, S., Chaowongdee, S., Roytrakul, S., & Sawwa, A. (2022). Analysis of proteomic changes in cassava cv. Kasetsart 50 caused by Sri Lankan cassava mosaic virus infection. BMC Plant Biology, 22(1), 573.