NEW ERA AGRICULTURE MAGAZINE

Mechanisms and Durability of Genetic Resistance in Plant Virus Disease Management

Gireesha D[1], Narsing Laxmi Prasanna[2], Kalpana Tilak[3], Apurba Das[4] and K. Gopika[5]

Abstract: -

Plant viruses are major agricultural pathogens and cause numerous economically significant plant diseases. Currently, there are limited effective control measures for viral infections; however, utilizing genetic resistance stands out as the most promising strategy. This approach often provides effective protection without incurring additional costs or labor during the growing season and without causing environmental harm. Sources of virus resistance have been identified for most crop species, and many resistant varieties are already commercially available and widely cultivated. Nonetheless, a deeper understanding of genetic resistance is still needed. This review focuses on three key areas that require further research. First, it examines the identification of resistance sources, highlighting the efforts of plant breeders and pathologists in germplasm screening and analyzing resistance traits specific to viruses. Second, it explores the molecular mechanisms underlying resistance, presenting case studies that detail how resistance functions at different stages of the viral infection cycle. Third, it discusses the durability of resistance on a global scale, analyzing factors that affect its longevity and how its stability can be predicted. The article concludes by exploring new technological and scientific opportunities emerging from recent advancements in this field.

Introduction:

Plant viruses are a leading cause of crop diseases, posing a major challenge due to the limited availability of effective control methods, making them among the most significant agricultural pathogens. Viruses are responsible for most newly emerging plant

Gireesha D[1], Narsing Laxmi Prasanna[2], Kalpana Tilak[3], Apurba Das[4] and K. Gopika[5]

[1]Assistant Professor, Department of Plant Pathology, School of Agriculture, SR University, Warangal, Telangana, India

[2]Ph.D. Research Scholar, Department of Plant Pathology, Navsari Agricultural University

[3]Assistant Professor, Department of Biotechnology, Chandigarh University

[4]Assistant Professor, Department of Plant Pathology, College of Sericulture, AAU, Assam

[5]Assistant Professor, Department of Plant Pathology, PJTSAU, Agricultural College, Warangal

diseases, with recent large-scale outbreaks including tospo viruses gemini viruses, Plum pox virus in stone fruit trees in the northern hemisphere, and Pepino mosaic virus in tomato crops across Europe and North America. Viral infections can significantly lower crop yields and impact food quality, with additional quality concerns arising from pesticide use aimed at controlling insect vectors. An alternative approach to combating viral diseases is the use of crop varieties that possess genetic resistance. These resistant cultivars carry heritable traits that can inhibit virus replication and spread, even under conditions favorable to infection. Genetic resistance offers several advantages, providing effective, cost-free protection during the growing season and being both environmentally sustainable and safe for consumers. Many crop species have access to virus-resistant traits, and numerous resistant cultivars have already gained commercial success. However, there are still several aspects of utilizing genetic resistance for the long-term control of viral diseases that require further investigation. This review focuses on 'natural' forms of resistance, while the development and application of transgenic resistance have been addressed in recent studies.

Breeding for resistance

The development of resistant cultivars that still produce high yields and excellent quality commodities remains a challenge for breeders. Plant breeders and plant pathologists have traditionally approached breeding for resistance in successive steps, including (i) screening germplasm collections to identify sources of resistance and characterising their phenotypes; (ii) studying the mode of inheritance and identifying genetic markers for marker-assisted selection (MAS); and (iii) introgressing resistance traits into elite cultivars and assessing performance of the new cultivars under pathogen challenge in the field. We will now consider these steps in detail, focusing on specific aspects of virus resistance.

Resistance mechanisms

For a plant to be susceptible to a virus, the pathogen must successfully navigate several stages: transmission from one plant to another, replication within the initially infected cells, movement to neighboring cells via plasmodesmata, systemic spread throughout the plant using the vascular system, and subsequent acquisition by vectors to perpetuate the infection cycle. Given the limited genome size of viruses, they rely heavily on both vector and plant host factors to complete these stages. These host factors must interact with viral proteins to facilitate the infection process,

meaning their absence or a change in their form that disrupts recognition by viral components can lead to resistance. This type of resistance is known as passive resistance, as it does not involve active defense responses by the plant. Genetically, such resistance is typically associated with recessive alleles of host susceptibility genes, where the dominant alleles would otherwise confer susceptibility. Conversely, plants can actively respond to viral infections through extensive metabolic changes, including the activation of various defense mechanisms. When these defense responses are effective against the virus, the plant exhibits active resistance, which is often governed by dominant resistance alleles.

In the following sections, we will explore case studies of both passive and active resistance, examining specific instances where these mechanisms interfere with different stages of the viral infection cycle. We will focus on key stages such as plant-to-plant transmission, intracellular virus replication, and systemic virus movement. Additionally, we will discuss the host genes responsible for resistance, as well as viral genes or sequences that determine the virus's ability or inability to infect resistant hosts (i.e., virulence and avirulence factors).

Resistance to plant-to-plant transmission

Primary infections of plant viruses typically originate from contaminated plant materials, such as seeds, via vector transmission from infected reservoir hosts, or through contact between damaged host tissues and contaminated soil or crop residues. Epidemiologically, around 18% of plant viruses are known to be transmitted through seeds in at least one of their host plants, while the vast majority—approximately 80%—rely on vectors for transmission. These vectors predominantly include insects but can also involve nematodes, fungi, and mites. Seed transmission involves intricate interactions between the virus and the physiological processes of two plant generations. It can occur through two main pathways: direct embryo infection after fertilization or indirect transmission via infected gametes. In the case of direct embryo infection, the virus must penetrate maternal tissues during a specific developmental window of the embryo, which may be influenced by environmental factors. Alternatively, the infection of gametes can facilitate viral transmission if the virus manages to infect meristematic tissues, a process often controlled by specific host factors. These two transmission mechanisms are not mutually exclusive, and many viruses likely utilize a combination of both pathways to achieve successful seed transmission.

Resistance to intracellular virus multiplication

Around 80% of plant viruses possess single-stranded, positive-sense RNA genomes. Their replication cycle in host cells typically starts with the release of the viral genome (desencapsidation) into the cytoplasm. This is followed by the synthesis of viral proteins using the host's cellular machinery and subsequent replication of the viral genome. Genome sequencing has revealed that positive-strand RNA viruses share certain core features, suggesting they employ similar strategies for replication and gene regulation. In fact, recent studies have identified several host proteins that facilitate viral translation and replication, which are common across different plant virus genera. Host susceptibility factors have been extensively studied in model organisms like Saccharomyces cerevisiae and Arabidopsis thaliana by analyzing large mutagenized populations. Insights gained from these artificially induced mutations have not only advanced the understanding of viral infection mechanisms but also hold potential for application in crop plants. Notably, two distinct plant RNA viruses, Brome mosaic virus (BMV) and Tomato bushy stunt virus (TBSV), have been shown to replicate in yeast cells, leveraging yeast resources for genome-wide mutant screening. For instance, research has demonstrated that a yeast protein, OLE1—a Δ9 fatty acid desaturase—is crucial for BMV replication. The absence of this enzyme disrupts membrane composition, thereby inhibiting viral replication. In Arabidopsis thaliana, studies on mutants tom1 and tom2A revealed that the replication of Tobacco mosaic virus (TMV) is hindered in the protoplasts of these mutant plants. The Tom1 and Tom2A genes encode transmembrane proteins that localize to the tonoplast and interact with both each other and the helicase domain of the viral replicase, playing a key role in the viral replication process. Further screening of A. thaliana mutants identified lsp1, in which the replication and gene expression of Turnip mosaic virus (TuMV) and Tobacco etch virus (TEV) are blocked. The lsp1 gene encodes the eukaryotic translation initiation factor (iso)4E (eIF[iso]4E), a member of the eIF4E family of translation factors, which has been implicated as a susceptibility factor for many plant viruses, in eIF4E-related genes have been shown to confer resistance to various viruses, especially within the Potyviridae family, whose genomes typically include a 3′-poly(A) tail and a 5′-end viral protein (VPg) covalently attached. The VPg protein often functions as an avirulence determinant in different virus-host interactions. The interaction between eIF4E and VPg is closely linked to the virus's ability to infect the host, influencing virus infectivity across numerous potyvirus-host systems.

Resistance to virus translocation and colonization of whole plants

After a virus has successfully multiplied in the initially infected cells, it needs to spread to neighboring mesophyll cells via plasmodesmata, a process known as cell-to-cell or local movement. During this phase, the virus replicates within each newly infected cell, resulting in a high concentration of viral particles that eventually enter the phloem sieve elements. Through the vascular system, the virus then undergoes long-distance transport to other parts of the plant. Most plant viruses produce movement proteins (MPs) that facilitate this process. MPs bind to and modify single-stranded RNA viral genomes, increasing the size exclusion limit of plasmodesmata in mature leaves. This allows the viral RNA to move between cells. MPs are essential for cell-to-cell movement, often working alongside other accessory proteins. In some viruses, MPs also play a role in systemic movement, aiding in the spread of the virus throughout the entire plant. Host factors are also crucial for the virus's local and systemic spread. These factors typically contribute to the plant's susceptibility; any alterations or the absence of these host factors may lead to resistance against viral movement. While specific details about these host factors remain limited, recent discoveries of proteins involved in the structure and function of plasmodesmata have provided new insights into their role in viral translocation.

Conclusions

Most sources of resistance to plant viruses have been identified through phenotype-driven screenings, although a vast pool of germplasm remains unexplored. To fully exploit these genetic resources, efficient high-throughput screening methods are essential. One promising strategy involves the use of genetically engineered viral genomes labeled with markers, which can aid in screening and phenotypic evaluation. For instance, Whitham et al. (1999) developed a modified strain of Tobacco etch virus (TEV) that expresses a herbicide resistance gene, allowing for the rapid identification of mutants with either increased susceptibility or enhanced resistance in large collections of Arabidopsis mutants. Integrating this approach with high-efficiency inoculation devices can further streamline the process. Phenotypic screenings often uncover quantitative resistance traits, which are typically controlled by multiple genes (polygenic). While these traits are often overlooked by breeders due to their complexity compared to monogenic or oligogenic traits, they represent a rich, untapped source of resistance alleles. The isolation and analysis of quantitative trait loci (QTL) could facilitate the development of resistant varieties by making these complex

traits more manageable. However, challenges remain, such as enhancing diagnostic assays for QTL detection and identifying genetic markers for marker-assisted selection (MAS) (Maule et al. 2007). Much of the molecular data on virus resistance has been derived from model species, but for practical applications in crop breeding, these findings need to be translated into agricultural contexts. This requires several key research activities, including the development of high-throughput genotypic screening methods like Eco TILLING (Nieto et al. 2006), expanding genomic data for major crop species, and creating platforms for efficient genetic analysis of large mutant collections, such as TILLING (Targeted Induced Local Lesions IN Genomes). These tools and approaches are pivotal in harnessing the full potential of genetic resistance to combat plant viral diseases.

References

1. Johansen, E., Edwards, M. C., & Hampton, R. O. (1994). Seed transmission of viruses—current perspectives. Annual Review of Phytopathology, 32, 363–386. doi:10.1146/ annurev.py.32.090194.002051.
2. Johnson, R. (1981). Durable resistance: definition of, genetic control and attainment in plant breeding. Phytopathology, 71, 567–568. doi:10.1094/Phyto-71-567.
3. Jovel, J., Walker, M., & Sanfacon, H. (2007). Recovery of Nicotiana benthamiana plants from a necrotic response induced by a nepovirus is associated with rna silencing but not with reduced virus titer. Journal of Virology, 81(22), 12285–12297. doi:10.1128/JVI.01192-07.
4. Jridi, C., Martin, J. F., Marie-Jeanne, V., Labonne, G., & Blanc, S. (2006). Distinct viral populations differentiate and evolve independently in a single perennial host plant. Journal of Virology, 80, 2349–2357. doi:10.1128/ JVI.80.5.2349-2357.2006.
5. Maule, A. J., Caranta, C., & Boulton, M. I. (2007). Sources of natural resistance to plant viruses: status and prospects. Molecular Plant Pathology, 8, 223–231. doi:10.1111/ j.1364-3703.2007.00386.x.
6. Mensah, C., Di Fonzo, C., & Wang, D. (2008). Inheritance of soybean aphid resistance in PI567541B and PI567598B. Crop Science, 48, 1759–1763. doi:10.2135/ cropsci2007.09.0535.
7. Milligan, S. B., Bodeau, J., Yaghoobi, J., Kaloshian, I., Zabel, P., & Williamson, V. M. (1998). The root

knot nematode resistance gene Mi from tomato is a member of the leucine zipper, nucleotide binding, leucine-rich repeat family of plant genes. The Plant Cell, 10, 1307–1319

8. Whitham, S. A., Yamamoto, M. L., & Carrington, J. C. (1999). Selectable viruses and altered susceptibility mutants in Arabidopsis thaliana. Proceedings of the National Academy of Sciences of the United States of America, 96, 772– 777. doi:10.1073/pnas.96.2.772.