

Biology and Genetic Composition of Negative-Stranded RNA Viruses in Plants

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Citation: Vazquez M (2024) Biology and Genetic Composition of Negative-Stranded RNA Viruses in Plants. Electronic J Biol, 20(4):1-2

Received date: July 22, 2024, Manuscript No. IPEJBIO-24-19808; **Editor assigned date:** July 25, 2024, PreQC No. IPEJBIO-24-19808 (PQ); **Reviewed date:** August 08, 2024, QC No. IPEJBIO-24-19808; **Revised date:** August 15, 2024, Manuscript No. IPEJBIO-24-19808 (R); **Published date:** August 22, 2024, DOI: 10.36648/1860-3122.20.4.128

Description

Negative-Stranded RNA Viruses (NSVs) are a diverse group of viruses that play significant roles in the pathology of both animals and plants. These viruses have single-stranded RNA (ssRNA) genomes, where the RNA is complementary to the viral messenger RNA (mRNA) and must be transcribed into a positive strand to produce proteins. While NSVs in animals, such as the influenza virus, are well-studied, the biology and genetic composition of negative-stranded RNA viruses in plants are relatively less explored. Plant NSVs represent a unique class of pathogens that can cause severe diseases in crops, leading to significant agricultural losses.

This essay explores the biology, genetic composition and mechanisms of infection of negative-stranded RNA viruses in plants. It also examines their interactions with host plants, the transmission of these viruses and potential management strategies. Understanding these viruses is important for developing effective strategies to control plant diseases and protect global food security.

Negative-stranded RNA viruses in plants

Negative-stranded RNA viruses in plants belong primarily to the order bunyavirales, which includes several virus families, such as tospoviridae and peribunyaviridae. The most notable plant NSVs are members of the *Tospovirus* genus, including species like Tomato Spotted Wilt Virus (TSWV) and Groundnut Bud Necrosis Virus (GBNV). These viruses are transmitted by thrips, small insects that act as vectors, facilitating the spread of infection across crops. The genomes of negative-stranded RNA viruses are typically segmented, meaning they are divided into several pieces of RNA. This segmentation allows for the exchange of genetic material during viral replication, increasing genetic diversity.

For example, the TSWV genome is divided into three segments: The Large (L), Medium (M) and Small (S) RNA segments. Each segment encodes specific viral proteins that are important for the replication cycle and infection process. The L segment encodes the viral RNA-dependent RNA polymerase (RdRp), which is esse

-ntial for transcribing the negative-strand RNA genome into mRNA and replicating the viral genome. The M segment encodes glycoproteins responsible for viral entry and movement within host cells. The S segment encodes proteins involved in the suppression of host immune responses and viral movement within the plant. This segmented genome structure is an attribute of many plant NSVs and contributes to their genetic flexibility, which can lead to the emergence of new viral strains. The replication cycle of negative-stranded RNA viruses in plants involves several key steps: Plant NSVs enter host cells through wounds created by vector insects, such as thrips. Once inside the cell, the virus particles are uncoated, releasing the viral RNA segments into the cytoplasm. The viral RNA polymerase transcribes the negative-strand RNA genome into positive-strand mRNA, which is then translated into viral proteins by the host cell's ribosomes. The viral RNA polymerase replicates the negative-strand genome to produce new RNA copies, which are packaged into new virions (virus particles). Newly synthesized viral proteins and RNA genomes are assembled into virions. These virions then exit the infected cell and spread to neighboring cells through plasmodesmata, small channels that connect plant cells. In the case of tospoviruses, the viral particles can also move systemically throughout the plant, infecting distant tissues.

Genetic composition and evolution

Negative-stranded RNA viruses exhibit a high degree of genetic variability due to the error-prone nature of their RNA polymerases which lack proofreading mechanisms. This results in frequent mutations, contributing to the rapid evolution of these viruses. Additionally, the segmented nature of their genomes allows for genetic reassertment a process where different viral strains can exchange RNA segments when co-infecting the same host cell. Reassortment can lead to the emergence of new viral strains with altered virulence, host range and transmission efficiency. The genetic diversity of plant NSVs poses a significant challenge for disease management, as new strains can overcome plant resistance genes or adapt to new host species. For



instance, TSWV has developed resistance-breaking strains that can infect previously resistant tomato varieties, highlighting the need for continuous monitoring and the development of new control strategies. Plant NSVs have evolved sophisticated mechanisms to evade host immune responses and establish successful infections. Plants have an innate immune system that detects viral invasion through Pattern Recognition Receptors (PRRs), which recognize Pathogen Associated Molecular Patterns (PAMPs). One such defense mechanism is RNA silencing, where small interfering RNAs (siRNAs) degrade viral RNA, limiting viral replication. To counteract these defenses, plant NSVs encode Viral Suppressors of RNA silencing (VSRs). For example, the NSs protein of tospoviruses is a potent suppressor of RNA silencing. It binds to siRNAs and prevents them from guiding the degradation of viral RNA, allowing the virus to replicate unchecked. This arms race between plant defense mechanisms and viral counter-defenses is a critical factor in the outcome of viral infections.