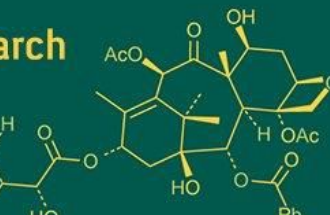
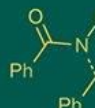


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(4): 21-28
www.biochemjournal.com
Received: 20-01-2026
Accepted: 24-02-2026

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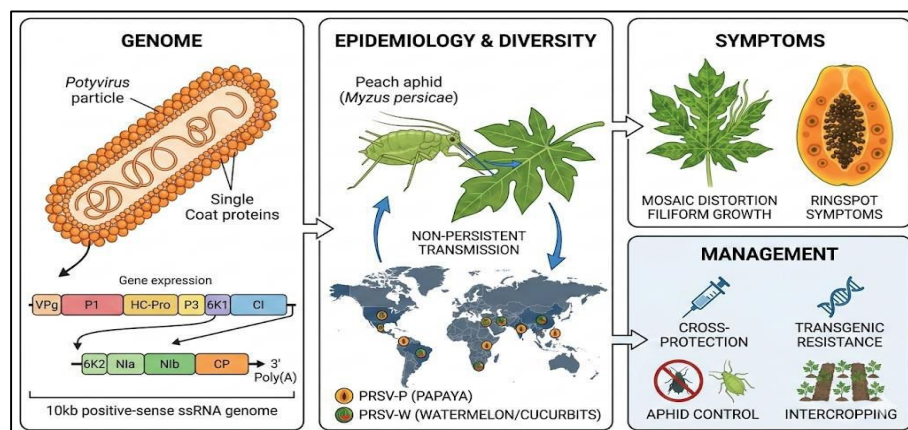
Papaya ringspot virus: Advances in biology, taxonomy, epidemiology, and control strategies

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DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i4a.8044>

Abstract

Papaya ringspot virus (PRSV) is one of the most devastating viral pathogens affecting papaya (*Carica papaya* L.) cultivation worldwide, causing severe yield losses and threatening the sustainability of papaya production in tropical and subtropical regions. Since its first recognition in the 1940s, PRSV has emerged as a major threat due to its rapid spread, wide host range, and efficient transmission by aphid vectors. Despite considerable advances in molecular characterization and the development of strategies such as cultural practices, vector management, resistant cultivars, and transgenic papaya, effective long-term control remains challenging due to the virus's high genetic variability, adaptability, and the limitations of current management approaches. Emerging molecular technologies such as RNA interference and genome editing offer promising avenues for future resistance development. This review synthesizes current knowledge on the taxonomy, genomic organization, genetic diversity, symptomatology, epidemiology, and management of PRSV, highlighting the need for integrated disease management strategies and continued molecular surveillance to mitigate the impact of this globally significant plant virus.



Keywords: *Papaya ringspot virus* (PRSV), taxonomy, genetic diversity, disease management

Introduction

Papaya (*Carica papaya* L.) is an economically important fruit crop cultivated widely in tropical and subtropical regions due to its high nutritional and commercial value. However, papaya production is severely constrained by several viral diseases, among which papaya ringspot disease caused by *Papaya ringspot virus* (PRSV) is considered the most destructive worldwide. The earliest description of the disease now recognised as papaya ringspot dates back to the early 1940s in Hawaii, where papaya growers reported unusual ring-like mottling on fruits and distortion of young leaves. Jensen (1949) and contemporaries described this disease as “papaya mosaic,” which was later renamed *papaya ringspot* due to its characteristic concentric ringspots on fruits [27]. The term “*Papaya ringspot virus*” (PRSV) was formally introduced following the identification of a viral agent transmissible through sap inoculation and aphids [2, 7, 27]. Initial identification of the disease was based on field observations, symptom expression, and transmission studies, as molecular diagnostic tools were not available at that time.

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Later, during the 1950s and 1960s, plants exhibiting similar disease symptoms were reported from several papaya-growing regions across Asia, Africa, and Latin America, indicating the rapid spread and global establishment of PRSV. During this period, early electron microscopy and serological tests revealed PRSV to be a filamentous virus, morphologically similar to members of the *potyvirus* group [44]. Subsequent mechanical inoculation experiments further confirmed that PRSV infects papaya and certain cucurbit hosts and is transmitted by several aphid species, particularly *Aphis gossypii* and *Myzus persicae*, in a non-persistent manner [19].

By the late 1950s, Capoor and Varma (1948) reported the occurrence of PRSV in India, marking one of the first documented cases of its spread beyond the Americas. By the late twentieth century, PRSV had emerged as the most serious viral constraint to papaya cultivation globally. In some regions, including Hawaii, disease outbreaks resulted in yield losses exceeding 90%, severely threatening commercial papaya production [20]. Similar epidemics were reported in India, Thailand, and the Philippines, where PRSV-induced fruit deformation and papaya unmarketable [51].

Advances in molecular virology during the 1990s enabled detailed characterization of the PRSV genome and revealed significant genetic variability among isolates from different geographical regions. Researchers, such as Gonsalves [20] have demonstrated that high aphid populations and the absence of resistant papaya cultivars drive PRSV epidemics. Cross-infection experiments also revealed that isolates from cucurbits could infect papaya, though symptom expression varied, suggesting intraspecies variation in virulence and host adaptation [56]. High mutation rates and recombination events contribute to the emergence of genetically diverse strains, complicating the development of durable resistance strategies. Such genetic diversity has been widely documented in PRSV populations from Asia, Africa, and South America, and it is thought to be a major factor contributing to resistance breakdown in transgenic papaya cultivars [43, 54]. Consequently, a comprehensive understanding of the virus's biology, genetic diversity, and epidemiology is essential for designing effective disease management approaches.

This review summarizes current knowledge on the taxonomy, genomic organization, genetic diversity, symptomatology, and epidemiology and management of *Papaya ringspot virus* and highlights the factors that influence its global distribution and persistence.

Taxonomic classification of *Papaya ringspot virus*

Papaya ringspot virus (PRSV) is taxonomically classified within the genus *Potyvirus*, family *Potyviridae*, which represents the largest family of plant RNA viruses. The species name *Papaya ringspot virus* is formally recognized in the year 1976 by the International Committee on Taxonomy of Viruses [24] with papaya (*Carica papaya* L.) designated as the principal host. Subsequent molecular phylogenetic analyses confirmed PRSV as a distinct species

within *Potyvirus*, clearly separated from other cucurbit-infecting potyviruses such as *Zucchini yellow mosaic virus* [25]. Then in 2023, the species designation for *Papaya ringspot virus* was formally revised to *Potyvirus papayanuli* [24] and the current taxonomic position is presented in Table 1.

According to the International Committee on Taxonomy of Viruses (ICTV), PRSV is distinguished from related potyviruses such as *Zucchini yellow mosaic virus* (ZYMV), *Watermelon mosaic virus* (WMV), and *Moroccan watermelon mosaic virus* (MWMV) based on sequence identity thresholds and host range [13, 51]. Comparative phylogenetic analyses suggest that PRSV forms part of the “PRSV cluster” of cucurbit-infecting potyviruses, a lineage that also includes WMVBV (*Wild Melon Vein Banding Virus*) and SuWMV (*Sudan Watermelon Mosaic Virus*). This cluster likely emerged in the Old World, with diversification events occurring around 3600 years ago, corresponding to ancient domestication and trade of cucurbits [13].

Table 1: Taxonomic position of *Papaya ringspot virus* [24]

Realm	<i>Riboviria</i>
Kingdom	<i>Orthornavirae</i>
Phylum	<i>Pisuviricota</i>
Class	<i>Stelpaviricetes</i>
Order	<i>Patatavirales</i>
Family	<i>Potyviridae</i>
Genus	<i>Potyvirus</i>
Species	<i>Potyvirus papayanuli</i>

Genomic organization of *Papaya ringspot virus*

The complete genome of PRSV was first sequenced by Yeh and Jan (1992), marking a pivotal milestone in understanding its molecular nature [56]. Their work revealed that PRSV contains a single-stranded positive-sense RNA genome of approximately 10,326 nucleotides, excluding the poly (A) tail at the 3' end. The genome encodes a large polyprotein of ~3344 amino acids, which is subsequently cleaved by three viral proteases (P1, HC-Pro, and NIa-Pro) into ten mature functional proteins: P1, HC-Pro, P3, 6K1, CI, 6K2, NIa-VPg, NIa-Pro, NIb, and CP (coat protein) [25, 51, 56]. The functional roles of these proteins are detailed in Table 2.

Structurally, the genome possesses untranslated regions (UTRs) at both ends, a 5'-noncoding region linked to a viral genome-linked protein (VPg) and a 3' polyadenylated tail (poly A tail), typical of *Potyviridae*. These UTRs play critical roles in replication initiation, translation enhancement, and interaction with host factors [49, 1]. Over time, comparative genomic studies revealed that PRSV exhibits high mutation rates and frequent recombination events, particularly within the P1, HC-Pro, and CP regions, contributing to its adaptability and geographical diversification [48].

Table 2: List of viral proteins and functions

Protein	Function
P1	Self-cleaving serine protease; influences host specificity.
HC-Pro	Helper component for aphid transmission and silencing suppression.
P3	Implicated in replication complex assembly.
6K1/6K2	Membrane-anchoring peptides involved in replication vesicle formation.
CI	RNA helicase facilitating movement and replication.
NIa-VPg	Primer for RNA synthesis; linked to replication initiation.
NIa-Pro	Main protease for polyprotein processing.
NIb	RNA-dependent RNA polymerase (RdRp); replication fidelity control.
CP	Coat protein for encapsidation, cell-to-cell movement, and vector interaction.

One of the defining characteristics of PRSV's molecular diversity lies in its biotype differentiation. Two biologically and molecularly distinct forms of PRSV are recognised globally, leading to the classification of two major biotypes: PRSV-P (papaya type) and PRSV-W (watermelon type). PRSV-P infects papaya as well as cucurbit hosts, whereas PRSV-W is restricted to cucurbitaceous plants and does not infect papaya [51]. Molecular comparisons in the late 1990s demonstrated that the two biotypes share high overall genome sequence similarity (over 85-90%) but differ in specific genomic regions, particularly within the coat protein gene and the helper component-proteinase (HC-Pro), which influence host specificity and aphid transmissibility [25]. PRSV-P and PRSV-W differ by discrete nucleotide and amino acid substitutions in critical regions, including P1, HC-Pro, P3, and the N-terminal domain of the coat protein, with these localized variations underpinning host adaptation and enabling PRSV-P to expand its host range [4, 34, 51].

Phylogenetic and recombination analyses indicate that PRSV-P likely evolved from a PRSV-W ancestor following the introduction of papaya from the Americas to Asia approximately 400-500 years ago [8]. More recent phylogenetic analyses have shown that PRSV-P and PRSV-

W do not form completely separate evolutionary lineages but instead cluster according to geographic origin, suggesting ongoing evolution and host-driven selection pressures [33].

Genetic diversity of *Papaya ringspot virus*

Comprehensive genome-wide comparisons have revealed substantial variation among PRSV isolates across regions. The first complete PRSV genome was sequenced from a PRSV isolate from Hawaii [56]. The coat protein (CP) gene, commonly used as a molecular marker, exhibits nucleotide divergence of up to 10% between isolates [35]. Analyses of isolates from Brazil, Cuba, Puerto Rico, India, and Taiwan have revealed strong geographic clustering, consistent with limited gene flow and region-specific evolution [34, 60]. High genetic variability in CP sequences has been reported within and between countries, including Brazil [32], India [39], Mexico [40], China [46], Thailand, Vietnam, and the Philippines [5]. In Mexico, the greatest PRSV variability occurred in regions with extensive papaya cultivation, likely linked to movement and exchange of plant genetic resources [40].

Table 3: Molecular characterization and sequencing studies of Papaya ringspot virus isolates from different geographic regions.

Sl. No.	Geographic origin of isolate(s)	Gene(s)/genomic region(s) targeted	Sequencing level	Year	Reference
1	Brazil	Coat protein (CP) gene	Partial sequencing	2002	Lima <i>et al.</i> [32]
2	China	Coat protein (CP) gene	Partial sequencing	2007	Wei <i>et al.</i> [53]
3	Hawaii	CP, HC-Pro, genome organization	Complete genome	2008	Tripathi <i>et al.</i> [51]
4	East Timor & Northern Australia	Whole genome (HTS)	Complete genome	2017	Maina <i>et al.</i> [33]
5	Cuba and American continent	Partial CP gene	Partial sequencing	2019	Cabrera Mederos <i>et al.</i> [6]
6	South India (Karnataka)	P1, HC-Pro, NIb-CP overlap	Partial sequencing	2019	Mishra <i>et al.</i> [37]
7	Bangladesh	Whole genome	Complete genome	2019	Hamim <i>et al.</i> [23]
8	Ecuador	Whole genome	Complete genome	2021	Medina-Salguero <i>et al.</i> [36]
9	Pakistan	Whole genome, CP, HC-Pro, P1	Complete genome	2021	Saleem <i>et al.</i> [48]
10	India (Karnataka)	Whole genome	Complete genome	2023	Premchand <i>et al.</i> [43]
11	Taiwan, Hawaii, Thailand, Mexico	CP gene (transgene-related)	Partial/comparative	2024	Yeh <i>et al.</i> [57]
12	India (Tamil Nadu)	Coat protein (CP) gene	Partial sequencing	2024	Mudnur <i>et al.</i> [39]

Collectively, these studies underscore the considerable genetic variability of PRSV populations across different geographic regions and highlight the role of mutation, recombination, and geographic isolation in shaping the evolutionary dynamics of the virus. A summary of studies on the sequencing and molecular characterization of PRSV isolates from various locations is presented in Table 3.

Symptomatology of *Papaya ringspot virus*

Early virological studies on *Papaya ringspot virus* (PRSV) primarily focused on symptom expression and field transmission. Infected papaya plants typically exhibit mosaic and chlorotic mottling on leaves, water-soaked rings

on fruits, and severe stunting, often leading to substantial yield losses. These characteristic symptoms were successfully reproduced through mechanical inoculation experiments, thereby confirming the viral etiology of the disease [7].

PRSV infection in papaya is associated with a wide range of symptoms that may vary depending on the stage of infection and environmental conditions. Initial symptoms generally appear as mild mosaic patterns and chlorotic mottling on young leaves, followed by blistering and distortion of leaf lamina. As the disease progresses, leaves may become narrow, filiform, or shoestring-like, often resembling symptoms caused by mite infestation. Petioles and the upper

stem frequently develop elongated dark green streaks accompanied by blistering symptoms. Under severe infection, fruits exhibit oily streaks and characteristic chlorotic ringspots, often accompanied by varying degrees of malformation and structural abnormalities. These symptoms considerably reduce fruit quality and marketability. When infection occurs during early stages of plant growth, severe stunting and substantial yield reduction are commonly observed [18, 21, 47].

PRSV can systemically infect hosts at any developmental stage [21]. Based on host range, the virus is classified into two major biotypes: PRSV-P (papaya type), which infects members of the families *Caricaceae*, *Cucurbitaceae*, and *Chenopodiaceae*, and PRSV-W (watermelon type) (previously designated as *Watermelon Mosaic Virus-I*), which infects cucurbitaceous hosts and *Chenopodiaceae* but

does not infect papaya. Both biotypes are transmitted non-persistently by aphid vectors [50].

Symptom expression differs between papaya and cucurbit hosts, as summarized in Table 4. In papaya, infection is typically characterised by prominent ringspots on fruit, along with severe leaf distortion and plant stunting. In contrast, cucurbit hosts generally exhibit foliar symptoms such as mosaic, mottling, and leaf deformation, while ringspot formation is rarely observed [33]. Depending on the time at which plants become infected and how widely the disease spreads, losses can reach up to 100%. Infected fruits may also show deformities and discoloration, further reducing their commercial value [16, 46]. This host-dependent symptom expression reflects differences in virus-host interactions and tissue tropism.

Table 4: Comparison of symptomatology between PRSV-P and PRSV-W strain

Biotype	Papaya (<i>Carica papaya</i>) PRSV-P	Cucurbits (melon, squash, cucumber) PRSV-W
Initial symptoms	Vein clearing, mild mosaic on young leaves	Vein clearing, mild mosaic
Leaf symptoms	Severe mosaic, blistering, shoestring, leaf distortion	Mosaic, leaf blistering, vein banding
Stem and petiole symptoms	Oily streaks, water-soaked lesions, necrosis	Mild streaking or absent
Fruit symptoms	Concentric ringspots, malformation, uneven ripening	Fruit deformation and mottling; ringspots absent
Growth effects	Severe stunting, reduced flowering, plant decline	Reduced vigour and yield
Diagnostic significance	Fruit ringspots are PRSV-P specific	Symptoms overlap with other cucurbit viruses

Biology and epidemiology of *Papaya ringspot virus*

Papaya ringspot virus (PRSV) is a filamentous plant virus whose particle, or virion, consists of a nucleocapsid forming a flexuous rod approximately 760-800 × 12 nm in size. The virions are filamentous and non-enveloped, comprising about 94.5% protein and 5.5% nucleic acid [51].

The virus is characterized by the cryptogram R/1, X/5.5, E/E, S/Ap, indicating that it possesses a single-stranded RNA genome (5.5% by weight), an elongated nucleocapsid structure, infection of seed-transmitted hosts, and aphid-mediated transmission [20]. The thermal inactivation point (TIP) of PRSV ranges from 54-56 °C, with a dilution end point of 10⁻³ and an *in vitro* longevity of about 3 hours, as reported by Conover (1962) [10]. Furthermore, purified PRSV virions exhibit a buoyant density of 1.32 g/cm³ in caesium chloride gradients [14].

In natural conditions, PRSV is primarily transmitted by several aphid species in a non-persistent manner. Studies by Yemewar and Mali (1980) demonstrated successful transmission of PRSV from papaya to papaya and also to *Cucumis sativus*, *Benincasa hispida*, and *Cucurbita maxima* through the aphid *Aphis gossypii* [58]. In non-persistent transmission, the virus does not circulate or replicate within the aphid vector but is retained briefly on the insect's stylet and rapidly lost after probing.

Aphid vectors such as *Aphis gossypii* and *Myzus persicae* can acquire the virus within seconds while probing infected plants and subsequently transmit it during short feeding periods on healthy hosts. Experimental observations indicate that an aphid feeding on an infected papaya plant for as little as 15 seconds may acquire the virus and initiate infection in a healthy plant [21, 51]. Therefore, The size and dynamics of aphid populations play a critical role in PRSV transmission and are strongly influenced by environmental conditions [3].

Several aphid species have been identified as efficient vectors of PRSV in different regions. In India, *Aphis gossypii*, *Aphis craccivora*, and *Aphis citricola* have been recognized as principal vectors, while in South Indian

papaya-growing regions *Myzus persicae* also contributes significantly to virus transmission [29, 30, 38]. Transmission efficiency studies using yellow funnel traps revealed *M. persicae* (56%) and *A. gossypii* (53%) as superior vectors compared to *A. craccivora* (38%), although the high field abundance and mobility of *A. gossypii* make it epidemiologically the most important vector [30].

Apart from vector-mediated transmission, PRSV can also spread through mechanical sap inoculation, which may occur during routine agricultural practices such as pruning, plant handling, and other crop management operations [25]. In contrast, seed transmission of *Papaya ringspot virus* (PRSV) appears to be minimal, with only 0.15% of symptomatic papaya plants reported to arise from infected seeds [5]. Although this pathway has been considered epidemiologically insignificant [19], subsequent studies have suggested the need to reassess seed transmissibility in potyviruses [17]. Notably, seed transmission of PRSV has been demonstrated in *Robinia pseudoacacia*, representing a rare but confirmed case [31].

Agricultural practices also significantly influence the epidemiology of PRSV. Continuous cropping, lack of crop rotation, mixed cultivation of papaya with cucurbit crops, and poor field sanitation can enhance disease incidence and facilitate virus spread. Host range studies have identified several plant species, including *Chenopodium amaranticolor*, *Cucumis sativus*, *Cucumis melo* var. *agrestis*, *Cucurbita pepo*, *Luffa cylindrica*, *Citrullus lanatus*, *Trichosanthes cucumerina*, *Cucumis melo*, *Lagenaria siceraria*, *Momordica charantia*, *Luffa acutangula* and *Carica papaya* as alternate hosts of aphids and *Papaya ringspot virus* (PRSV). These plants function as virus reservoirs, supporting aphid populations and enabling the persistence of PRSV between cropping cycles [12]. Notably, the weed *Momordica charantia* (cerasee) was reported as a natural host of PRSV in papaya orchards in western Jamaica, where infection was confirmed through DAS-ELISA and RT-PCR assays [9]. The presence of such

alternative hosts allows the virus to survive between cropping seasons and maintain a continuous disease cycle in the field^[11].

Warm temperatures combined with moderate humidity favour rapid aphid multiplication, thereby increasing the likelihood of virus spread and leading to severe disease outbreaks in tropical and subtropical regions^[45]. Seasonal variations in PRSV incidence are therefore closely associated with aphid activity and cropping patterns, with disease outbreaks generally peaking during warmer months when aphid populations are highest, while reduced transmission occurs during cooler or dry periods^[23]. Understanding these seasonal patterns is essential for planning effective disease management strategies.

Papaya ringspot disease management

Papaya ringspot virus remains one of the most destructive viral pathogens affecting papaya cultivation worldwide due to its rapid aphid-mediated transmission, considerable genetic variability, and adaptability to diverse environmental conditions. Due to the absence of curative treatments for viral diseases in plants and the rapid transmission of PRSV by aphids in a non-persistent manner, effective management relies primarily on preventive and integrated strategies. Numerous studies have investigated cultural practices, vector management, host resistance, transgenic technologies, cross-protection, and emerging molecular approaches to mitigate PRSV epidemics and sustain papaya production.

Cultural practices for PRSD management

Cultural practices play an essential role in limiting the spread of PRSV within papaya plantations. Early detection and removal (rouging) of infected plants can substantially reduce primary inoculum sources and prevent secondary spread of the virus within orchards^[38, 51]. Epidemiological studies indicate that systematic rouging of infected plants can effectively slow disease progression and reduce the incidence of Papaya ringspot virus in commercial papaya systems. Additionally, planting time plays a critical role in disease dynamics, with early June or late March planting identified as an effective cultural strategy, whereas planting during September-January should be avoided due to increased aphid vector activity and the associated risk of infection^[42].

Maintaining strict field sanitation is another important component of PRSV management. Removal of volunteer papaya plants and weed hosts reduces virus reservoirs that may harbour PRSV and sustain aphid populations between cropping seasons^[4]. Avoiding intercropping papaya with cucurbit crops is also recommended because cucurbits such as cucumber and squash can serve as alternative hosts of PRSV and facilitate virus persistence in agricultural ecosystems^[51].

Reflective plastic mulches have been widely studied as a strategy to reduce vector activity. Reflective surfaces alter ultraviolet light reflection patterns and interfere with aphid host-finding behaviour, thereby reducing aphid landing rates on papaya plants^[41]. Field experiments have demonstrated that reflective mulches can delay the onset of PRSV symptoms and significantly reduce early-season infection in papaya plantations.

Vector management strategies

Papaya ringspot virus is transmitted in a non-persistent manner by several aphid species, with *Aphis gossypii* and *Myzus persicae*, enabling rapid acquisition and transmission within seconds of probing. Consequently, conventional insecticide applications are generally ineffective in preventing primary infection, as virus transmission often occurs before the insecticide can act^[20]. Although insecticides may reduce aphid population densities, their role in limiting virus spread remains constrained. Nevertheless, some field studies have reported that intensive spray schedules integrating insecticides and micronutrients (8-12 applications at 20-30 day intervals) can suppress Papaya ringspot disease, delay symptom development, and enhance plant growth, yield, and economic returns under specific conditions^[42].

Alternative approaches such as mineral oil sprays have been investigated as a means to reduce virus transmission. Mineral oils interfere with virus acquisition and inoculation by forming a thin protective film on plant surfaces that disrupts virus attachment to aphid stylets^[41, 52]. Several studies have shown that mineral oil applications can reduce transmission efficiency of aphid-borne viruses, including PRSV, under field conditions.

Barrier cropping using *Zea mays* and reflective mulching are additional vector management strategies that can limit aphid colonization and reduce virus spread. These approaches alter aphid movement patterns and reduce the likelihood of virus transmission during early crop growth stages^[52].

Breeding for host resistance

The development of resistant cultivars remains one of the most sustainable strategies for controlling PRSV. However, conventional breeding programs have achieved limited success due to the narrow genetic diversity of cultivated papaya (*Carica papaya*) and the absence of strong natural resistance sources within the species. Resistance to PRSV has been identified in related wild relatives such as *Vasconcellea* species; however, transferring this resistance into cultivated papaya through conventional breeding is constrained by genetic incompatibility and reproductive barriers^[55]. Despite these limitations, efforts continue to identify resistant germplasm and develop interspecific hybrids to enable gene introgression. Recent molecular studies have revealed candidate resistance genes, including those encoding proteinase inhibitors, nucleotide-binding site-leucine-rich repeat (NBS-LRR) proteins, and serine/threonine protein kinases (STKs), in *Vasconcellea pubescens* and *Cucumis metuliferus*. Notably, the identification of an STK-encoding resistance gene in *V. pubescens* and its ortholog in *C. papaya* suggests significant potential for leveraging orthologous genes in developing PRSV-resistant papaya through advanced breeding and biotechnological strategies^[22].

Transgenic-Mediated Host resistance

The development of transgenic papaya cultivars expressing the PRSV coat protein gene represents one of the most successful examples of pathogen-derived resistance in plants.

This strategy involves introducing viral coat protein genes into papaya plants to interfere with virus replication and movement, thereby conferring resistance to PRSV infection. Transgenic papaya cultivars such as 'Rainbow' and

'SunUp', developed in Hawaii, exhibited high levels of resistance to PRSV and were successfully deployed in commercial production systems. Field evaluations demonstrated that these cultivars maintained stable resistance under natural infection conditions and played a crucial role in restoring papaya production in regions severely affected by PRSV epidemics^[15].

The successful deployment of transgenic papaya has been widely recognized as a landmark achievement in plant biotechnology and virus disease management. Recent studies have also examined the long-term durability of transgenic resistance and its effectiveness against diverse PRSV isolates.

Virus cross-protection approaches

Cross-protection involves the use of mild virus strains to protect plants against infection by more virulent strains of the same virus. This strategy has been explored as a potential management tool for PRSV in several studies. Freitas and Rezende (2008) demonstrated that mild PRSV isolates could reduce symptom severity and delay disease progression when papaya plants were subsequently challenged with severe virus strains^[16].

Although cross-protection has shown promising results under experimental conditions, large-scale implementation remains limited because of concerns regarding strain stability and the potential emergence of more aggressive virus variants.

Emerging molecular approaches

Recent advances in plant biotechnology have introduced new possibilities for developing durable resistance to PRSV. RNA interference (RNAi) technology has been widely investigated as a strategy for silencing viral genes involved in replication and movement, thereby providing resistance against PRSV infection^[28].

Genome editing technologies such as CRISPR/Cas systems have also emerged as promising tools for engineering virus resistance in crops. These technologies allow precise modification of host susceptibility genes or viral genomic sequences and may provide new opportunities for developing PRSV-resistant papaya cultivars^[59]. In addition, recent genomic studies have revealed considerable genetic variability among PRSV isolates, highlighting the importance of molecular approaches for developing durable resistance strategies^[48].

Integrated approaches to PRSD management

Because of the complex epidemiology of PRSV, no single control strategy is sufficient to effectively manage the disease. Integrated disease management approaches combining cultural practices, vector monitoring, resistant cultivars, and biotechnology-based solutions are therefore recommended. Continuous surveillance of virus populations, early detection of infected plants, and timely implementation of preventive management strategies can significantly reduce PRSV incidence and improve papaya productivity in virus-prone regions^[4, 51].

Future Prospects

Future research on *Papaya ringspot virus* (PRSV) should focus on developing durable and sustainable management strategies by integrating advances in molecular biology, genomics, and plant breeding. Comprehensive genomic

surveillance of PRSV populations across different geographic regions is essential to understand virus evolution, recombination, and the emergence of new virulent strains. Identification and utilization of novel resistance sources from wild relatives such as *Vasconcellea* species may provide new opportunities for breeding PRSV-resistant papaya cultivars. In addition, emerging biotechnological approaches including RNA interference (RNAi), CRISPR/Cas-based genome editing, and other gene-silencing technologies offer promising avenues for engineering broad-spectrum and long-lasting resistance. Improved understanding of virus-host interactions, vector ecology, and environmental influences on disease dynamics will further enhance predictive epidemiology and targeted management strategies. Integrating these advances with effective cultural practices and continuous monitoring of virus populations will be critical for ensuring sustainable papaya production and mitigating the long-term impact of PRSV.

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